Cervical cancer is the second most common cancer among women in the world. Approximately 450,000 new cases are diagnosed each year (World Health Organisation, 1996). Three quarters of these cases occur in the developing world. In Central and South America, parts of India and Sub-Saharan Africa, the incidence rate is between 3 and 5 times as high as in western Europe (Parkin, 1992). However, no form of cancer better documents the remarkable effects of early cancer diagnosis and curative therapy on the mortality rate than the cancer of the uterine cervix. In most developed countries, the death rate from cervical cancer has dropped remarkably. This is because of the fact that half of the invasive cancers are cured by effective therapy and most lesions are discovered while in situ and amenable to eradication by timely and appropriate treatment. Credit for these dramatic gains goes largely to the effectiveness of papanicolaou cytologic test in detecting cervical carcinoma during its incipency and to the accessibility of the cervix to colposcopy and biopsy. Although much is known about the natural history of cervical cancer than any other form of cancer, still it is difficult to pin point a single factor which may be involved in the genesis of this cancer. As in other forms of cancers, carcinogenesis of cervix is multifactorial. Various risk factors which are considered to be important for cervical cancer are age at first intercourse, number of sexual partners etc. The epidemiology of cervical cancer strongly suggests sexual transmission of an oncogenic agent most probably a virus. Although Herpes simplex virus type II (HSV II) was once suspected, current studies indicate human papilloma virus as an important factor in cervical oncogenesis. Besides that some oncogenes and antioncogenes occur in concert with HPV(s) for the genesis of this cancer and amongst these p53 and retinoblastoma gene are considered to be most important.

The present study was initiated to find out the role of possible factors in the genesis of cervical cancer with particular reference to incidence of HPVs, level of expression of p53 as well as chromosomal instability.

The data, thus, collected have indicated a high prevalence of HPVs predominantly HPV 16 followed by HPV 18. Over expression of p53 and increased baseline value of sister chromatid exchanges were detected in patients.

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