The current study was undertaken with the following objectives to:

- look for various risk factors responsible for the cancer of uterine cervix,
- determine the incidence of HPV 16, 18 and 11 in patients with cervical abnormalities as well as with cervical cancer,
- study the frequency of spontaneous chromosomal fragility in patients with cervical cancer,
- study the level of p53 protein in the serum of patients with cervical cancer by using Pan ELISA assay.

Epidemiological data collected from the patients showed that age of patients ranged from 30-70 years. All the patients were sexually active and 98% of them were multiparous. Majority of the patients (44%) got married between the age of 15-17 years. Seventy percent of the patients belonged to rural areas with lower socioeconomic status.

Most of the patients (66%) presented themselves at stage III of cervical cancer indicating lack of early detection or screening by pap smear.

Cytogenetic analysis of the lymphocytes of 10 patients and five controls showed a statistically significant difference in the SCE frequency in patients (4.48±2.72) as compared to control subjects (2.48±1.60).

The incidence of different HPV types i.e. 11, 16, 18 was studied in 42 patients ranging from minor abnormalities of cervix, dysplasia and CIN to invasive cervical cancer by using dot blot hybridization. The dot blot hybridization revealed the presence of HPV DNA in 70% of cases with invasive cervical cancer and in 63.6% of cases with cervical abnormalities, dysplasia and CIN.

The incidence of HPV 16 was 70% in invasive cancer, while it was 45.4% in cases with cervical abnormalities. However the incidence of HPV 11 was higher in cases with cervical abnormalities (36.7%) in comparison to those with invasive cancer (15%). HPV 18 was found in 30% of invasive cases, while no
signal was obtained in benign lesions or cervical abnormalities.

The incidence of HPV was also studied by polymerase chain reaction (PCR) in some of the samples. In invasive cases 10 out of 13 samples (76.9%) were found to be positive by PCR and some of these samples were detected negative for HPV by using dot blot hybridization. However in cases with benign lesions and cervical abnormalities only 2 samples out of 7 tested were positive for HPV DNA.

Overexpression of p53 using Pan ELISA Assay was detected in 8/13 (61.5%) serum samples of patients with cervical cancer. The highest value of p53 was observed in carcinoma in situ. No correlation was observed between the value of p53 and progression of disease. However the value of p53 was found to decrease with increase in the age of the patients. The correlation of p53 overexpression and status of HPV revealed that both the factors can function together or independently towards the genesis of cervical cancer.

The present study indicated a high genomic instability in cervical cancer patients as shown by the sister chromatid exchange analysis. A high incidence of HPV was observed in cervical cancer patients and HPV-16 as the dominant type, followed by HPV-18 and 11. High level of p53 protein revealed the involvement of tumour suppressor gene in cervical cancer. However, the study of p53 status in relation to HPV infection did not reveal any definite pattern suggesting that alteration in p53 expression and HPV infection may or may not act in concert towards carcinogenesis of cervix.