INTRODUCTION

Cervical cancer was once a leading cause of cancer death in the world. Now, invasive cervical cancers are relatively uncommon. This change is probably mostly due to effective identification and eradication of cancer precursor lesions. Potentially cancerous precursor lesions found on the uterine cervix are referred to as cervical intraepithelial neoplasia, or CIN (Schlecht et al, 2002).

Persistent human papillomavirus (HPV) infection is requisite to the development of cervical neoplasia .Of more than 100 serotypes of HPVs approximately 40 may be involved in anogenital area. Approximately 15 HPV serotypes are considered oncogenic, causing virtually all cases of cervical cancer. HPV 16 alone accounts for over 50% of cancers and HPV 18 is responsible for an additional 10% (Dalstein et al, 2003).

It is well established that invasive carcinoma of cervix is preceeded by a precursor lesion that morphologically resembles the adjacent invasive squamous carcinoma. This lesion is termed “carcinoma in situ” (CIS). However, CIS itself is preceeded by a spectrum of lesions with varying degrees of abnormality. The term “dysplasia” was introduced to refer to this spectrum of progressive cervical abnormality from normal epithelium to CIS. Histologically, dysplasia is sub-classified into mild, moderate, or severe based on the extent to which the cervical epithelium is involved with abnormal cells: 1/3rd, 2/3rd or full thickness respectively (Molano et al, 2003).

Dysplasia refers to loss of the normal cytoplasmic differentiation or maturation that occurs with progression from the basal cell to the superficial keratinocyte in the cervical epithelium. The area of development of dysplasia and squamous cell cervical cancers is at the junction of the squamous and columnar epithelium and this area is evidently most susceptible to viral infection. The location of this junction, termed the transformation zone, is dynamic and responds to changes in vaginal pH in response to fluctuating estrogen levels (Wright et al, 1995).

The term "Cervical intraepithelial neoplasia" (CIN) was introduced and implied the concept that precursor lesion to squamous cell carcinoma represents a single, continuous disease process. CIN nomenclature for histology is more specific to the cervix than the general term "dysplasia," and makes clear the pre-invasive nature of lesions. The CIN nomenclature divides cervical cancer precursors into CIN1, CIN2, and CIN3, corresponding to mild, moderate and severe dysplasia / carcinoma in situ (Peyton et al, 2001).

There are usually no symptoms or signs of CIN, and the diagnosis is most often based on biopsy findings following an abnormal routine cervical cytology smear. As recommended by the American College of Obstetricians and Gynecologist, all women who have been sexually active or reached age 18 should have a pelvic and cytological examination at least once a year. After three or more consecutive, satisfactory, normal annual examinations, the screening interval may be extended in selected low-risk patients. However, annual screening should be continued in women with any risk factor for CIN (Coppleson, 1992).

Cytological abnormalities can be diagnosed by examining the cervical Papanicolaou smear (Pap smear). All abnormal Pap smears require further evaluation, such as colposcopy ,visual inspection of the cervix, repeated cervical cytology, staining with Lugol's solution (Schiller test) or toluidine blue, or diagnostic conization. The objective is to exclude the presence of invasive carcinoma and to determine the degree and extent of any CIN lesion (Christine, 2003).

Following expert colposcopic evaluation, diagnostic cone biopsy of the cervix is indicated if colposcopy is unsatisfactory, for a high –grade cervical cytology smear, if the lesion extends into the cervical canal beyond the view afforded by the colposcope, cytological diagnosis inconsistent with histological diagnosis based on directed biopsy findings, if adenocarcinoma in situ is suspected, or if microinvasive carcinoma is suspected (Andrews et al, 1998).

Ideally, the natural immune response would be powerful enough to eradicate any low-grade CIN or tissue abnormalities. Currently, there is no treatment per se for CIN I, which either resolves or progresses to CIN II, which is treated. If CIN I does not resolve but instead progresses, or is detected at CIN stage II or III, treatment is needed to prevent the development of invasive disease (Leslie, 1996).

CIN lesions may be treated on an outpatient or inpatient basis. Outpatient techniques include electrocautery, cryosurgery, laser vaporization, cold coagulators and excision and loop electrosurgical excision procedure (LEEP); inpatient techniques include cone biopsy or cervical (cold knife) conization and simple total hysterectomy (Philip et al, 1997).