A LONGITUDINAL STUDY OF CERVICAL DYSKARYOSIS DURING PREGNANCY

by

MAMTA SINGH,* B.Sc., M.D., D.C.P.

RAJ BAVEJA,** M.S., D.G.O., Ph.D.

RADHA JINA,*** M.S., D.G.O.

and

G. SINGH,**** M.D., M.A.M.S.

Introduction

Dysplastic changes in the cervical epithelium during pregnancy is now a well established fact. It is observed that during pregnancy the preexisting dysplasia of cervix gets flared up due to the effect of certain hormones whose levels are elevated during pregnancy (Wahi *et al*, 1969). The present study was undertaken to observe the dyskaryotic changes during pregnancy in cases with cervical erosion. A cohort of cases was followed to study the regression among these changes after delivery and puerperium.

Material and Methods

The cervical scrape smears were taken by means of Ayre's spatula and stained by standard Papanicolaou's (1954) technique. The criteria for cytological analysis were those described by Papinicolaou (1954), Watchel (1969), Wahi *et al* 1969), W.H.O. report (1973) and Novak (1974). The cases were classified into 7 groups: normal, chronic cervicitis, dysplasia of mild, moderate and severe grades, carcinoma in situ and invasive carcinoma (Singh *et al*, 1976).

Cervical punch biopsy was done in cases with smears, suggestive or conclusive of dyskaryosis. A follow-up study was done in cases with severe dysplasia or neoplasia in order to have a close watch on the progress of the lesion. Surgical interference was done in case of neoplasia, if any, along with termination of pregnancy.

Observations

Table 1 shows cytological findings in women with or without pregnancy. It reveals dysplasia in 26.9 per cent non-pregnant and 40.5 per cent in pregnant women. The higher frequency of dysplasia in pregnant group was statistically highly significant ($p < 0.001$). In the present series, group of mild dysplasia was observed in 59 (29.5%), moderate in 21 (10.5%) and 1 (0.5%) of severe dysplasia. No case of neoplasia was found. The women with dysplasias were followed cytologically during antenatal and puerperium for progression or regression of cervical atypia.

*Lecturer in Pathology.

**Professor and Head, Deptt. of Obstetrics and Gynaecology.

***Lecturer in Obstetrics and Gynaecology.

****Reader in Social and Preventive Medicine, M.L.N. Medical College, Allahabad-211 001.

Accepted for publication on 2-1-79.

TABLE I
Showing Cytological Distribution of Cases

Group of patients	Normal	Chronic cervicitis	Mild dysplasia	Moderate dysplasia	Severe dysplasia	Carcinoma in situ	In vasive Carcinoma	Total
Non-pregnant women	130 (30.6)	173 (40.75)	70 (16.5)	30 (7.06)	14 (3.3)	5 (1.17)	3 (.734)	425 (68.00)
Pregnant women	72 (36)	47 (23.5)	59 (29.5)	21 (10.5)	1 (0.05)	—	—	200 (32.00)
Total	202 (32.3)	220 (35.2)	129 (20.6)	51 (3.1)	15 (2.4)	5 (0.8)	3 (0.5)	625 (100.00)

Note: Figures in parantheses denote percentage.
$X^2 = 20.62$, $P < 0.001$.

During follow-up, 1 case of severe dysplasia (Photomicrograph-1) turned malignant (Photomicrograph-2) and histologically diagnosed as carcinoma in situ. One case each of mild and moderate dysplasia progressed to severe type during pregnancy. It was observed that most of the cases (99.5%) of cervical atypia reverted back to normal or milder forms of dysplasia during the puerperium. The subjects delivered safely without any complications. The case of severe dysplasia which progressed to carcinoma in situ was treated surgically and pregnancy was terminated.

Discussion

It is well known that cervical scrapings are not diagnostic of erosion cervix uteri since the abnormality is one of the atypical localization of epithelium rather than that of the cell character. But the importance lies in the differentiation of simple erosion from a malignant one (Watchel, 1969). In the present series, dysplasia was significantly higher among pregnant than non-pregnant women ($p < 0.001$).

In the general population, prevalence of dysplasia varies between 1.2 to 3.1 per cent (Murphy, 1950; Gusberg, 1951; Lapid and Goldberger, 1951; Mackay, 1959). While in erosion of cervix uteri cervical dysplasia is reported to be much higher, according to Rao *et al* (1973) and Gulati and Chandra (1973) it was observed to be 10.9 and 17.5 per cent respectively. Wahi (1969) also reported that maximum (65.3%) cases of cervical dysplasia were from erosion cervix uteri group. Pundal (1959) observed that epithelial atypia of cervix uteri was more frequent in pregnant women. In this study, the higher percentage of dysplasia among pregnant women might be due to co-existence of both the factors.

The dysplastic changes of cervix uteri are further exaggerated by prolonged influence of pregnancy on sensitive cervical epithelium, which may continue during lactation or successive pregnancies where the epithelial atypia does not get any time to regress (Epperson *et al*, 1951; Wahi, 1969). As these dyskaryotic changes in cervical epithelium are due to hormonal influence of pregnancy, these were followed till delivery. It was found that moderate to severe dysplasia cases regressed back to normal or to milder form during puerperium. MacLaren (1969) also reported similar observation add ng that cervival atypia at times progresses to malignancy during antenatal period as happened with 1 case in our study. This substantiates the role of cytological follow up which is vital during antenatal and postnatal care. The histopathological examination is not practical as in cone biopsy there is risk of haemorrhage and abortion, while in cervical punch biopsy the lesion may be missed.

It can be infered from the present study that the waiting policy is safe and permits normal delivery in the hospital as suspect smears (dyskaryosis), during follow up tests may revert back to normalcy or milder forms. But a regular cytological examination is essential as few of them may become positive for malignancy and demand surgical interference. Considering the high risk of cervical dyskaryosis during pregnancy a thorough cytological follow up must be included in antenatal care schedule.

References

1. Epperson, J. W. W., Helman, L. M., Galvin, G. A. and Busby, T.: Am. J. Obstet. Gynec. **61**: 50, 1951.
2. Gulati, N. and Chandra, K.: J. Obstet. Gynec. India. **23**: 347, 1973.
3. Gusberg, S. B.: Am. J. Obstet. Gynec. **61**: 276, 1951.
4. Lapid, L. S. and Goldberger, M. A.: Am. J. Obstet. Gynec. **61**: 1324, 1951.
5. MacLaren, H. C., Brit. Med. J. **1**, 216, 216, 1969.
6. Mckay, D. G.: Obstet. Gynec. **13**: 2, 1959.
7. Murphy, E. J. Am. J. Obstet. Gynec. **59**: 389, 1950.
8. Novak, E. R. and Woodruff, J. D.: Novak's Gynecologic and Obstetric Pathology with Clinical and Endocrinal relations, 7th Edn. W. B. Saunders Co. Philadelphia & London, 1974.
9. Pundal, J. P.: Acta Cytol. **2**: 353, 1959.
10. Papanicolaou, G. N.: Am. J. Obstet. Gynec. **85**: 458, 1954.
11. Rao, A., Krishna, V. and Purandare, B. N.: J. Obstet Gynec. India. **25**: 3, 315, 1973.
12. Singh, M., Baveja, R., Singh, G. and Mehrotra, T. N.: Indian J. Med. Res. **64**: 12, 1783-1785, 1976.
13. Wahi, P. N., Luthra, U. and Mali, S.: Indian J. Med. Res. **57**: 617, 1969.
14. Wachtal, E. G.: Exfoliative Cytology in Gynaecological Practice, 2nd. Edn. Butterworths, 1969.
15. W. H. O.: Cytology of the Female genital tract, report no. 8, 1973.

See Figs. on Art Paper II