STUDY OF GLYCOGEN AND ITS SIGNIFICANCE IN ENDOMETRIUM IN CASES OF PRIMARY STERILITY

by

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Introduction

It has been reported that abnormal glycogen content of endometrium throws sufficient light on any abnormality in neurohumoral-endometrium axis, as the glycogen content of endometrium is ovarian steroid dependent (Gregoive *et al*, 1973). Detection and demonstration of glycogen in endometrium by histochemical methods in primary sterility cases have been reported (Zondek and Stein, 1940; Alkinson and Engle, 1947; McKay *et al*, 1956; Tyagi *et al*, 1977). These authors have also found a positive correlation between primary sterility and amount of glycogen present in endometrium.

Therefore, a planned study was undertaken in the department of Pathology to evaluate the role of glycogen, thus hormonal status, in causation of primary sterility.

Material and Methods

The present work is a retrospective as well prospective one, based on the study of 250 endometrial biopsy obtained from primary sterility cases, at Mahatma Gandhi Institute of Medical Sciences, Sevagram over a period of 3 years, from 1978-80, both the years inclusive.

The clinical records were thoroughly reviewed and pertinent findings were noted. All the patients had undergone a preliminary investigations. Semen analysis of male and vaginal examination of female partners were carried out to exclude any pathology of the genital tract, if present.

Two sections were prepared from each paraffin block. One for the routine stain and another for periodic-acid-schiff's staining (Pearse, 1968). All the 250 sections in routine stain i.e. haematoxylin-eosin were screened while P.A.S. staining was done only in 189 sections of proliferative and secretory endometrium. The scoring of glycogen in glands and stroma of endometrium was done according to Arzac and Blanchet (1948).

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Observations

In the present study, secretory endometrium was found in maximum numbers of 140 (56%) cases, followed by proliferative in 49 (19.6%) cases, hormonal imbalance in 44 (17.6%) cases and tubercular endometritis in 17 (6.8%) cases.

On analysing 140 secretory endometrium 40 (28.6%) were found to be in early phase, while 100 (71.4%) were in late secretory phase. Out of 49 proliferative cases, maximum number of 42 (85.7%) cases were in late proliferative phase and only 4 (8.2%) and 3 (6.1%) cases were in hypoplastic proliferative and early proliferative phase respectively.

Analysis of periodic Acid-Schiff's Scoring was done. The late secretory endometrium showed nil to little glycogen in glands in 37 (26.4%) cases and in stroma in 85 (60.7%) cases, moderate to good amount in glands in 63 (45%) cases and in stroma in only 15 (10.7%) cases of late secretory phase. Similarly, early secretory endometrium showed, nil to little amount of glycogen in glands in 9 (6.4%) cases and in stroma in 18 (12.8%) cases, moderate to good amount in glands in 31 (22.1%) cases and in stroma in 22 (15.8%) cases.

All the 4 (8.2%) hypoplastic proliferative cases showed nil to little amount of glycogen in stroma and glands. Similar finding was observed with the glands and stroma of all the 3 (6.1%) endometrium with early proliferative phase. Out of 42 late proliferative endometrium nil to little glycogen was found in glands in 39 (79.6%) cases and in stroma in 33 (67.3%) cases and moderate to good amount of glycogen was seen in glands in only 3 (6.1%) cases and in stroma in 9 (18.3%) cases.

Discussion

Genital tract glycogen is unique in that unlike muscular glycogen, it is unaffected by either carbohydrate intake or exercises, but is controlled by ovarian steroids (Gregoive *et al,* 1973). It has been reported that oestrogen causes an increase in uterine phosphate and alkaline phosphatase (Atkinson and Engle, 1947). An inverse correlation of alkaline phosphatase with glycogen has also been reported by (Hughes *et al* 1963). Demonstration of alkaline phosphatase in preovulatory phase and accumulation of glycogen in ovulatory and postovulatory phase fits well into the phosphorrylation theory of glycogenesis (Tyagi *et al,* 1977).

Out of 189 cases, 140 cases were found 'in phase', but most of them showing disparity in distribution of glycogen. This defect, specially, was seen with the stroma in secretary phase. Similarly, Baweja *et al* (1972) in their series reported impaired distribution of glycogen in late secretory phase. Further it has also been reported that, reduced endometrial glycogen is an important contributing factor to sterility (Zondek and Stein, 1940). Hughes *et al* (1950) stressed on the deficient glycogenic property as the evidence of poorly prepared endometrium for the implantation of fertilized ovum. Moreover, decreased glycogen is also significant because of its association with lowered glucose content of the uterine fluid and cervical mucous, which affects the sperm migration (Chadha *et al,* 1980).

Out of 42 late proliferative endometrium, moderate to good amount of glycogen was seen in stroma in only 9 (18.3%) cases. Thus glycogen is present in abundance when it is not needed, again an impaired distribution of glycogen in endometrium.

Thus we believe that the presence of glycogen in appropriate amount, in particular period of development of endometrium is essential for fertility.

The present study suggests that the determination and demonstration of glycogen in endometrium, alongwith its histomorphology, should be carried out in cases of primary sterility to ascertain the content of glycogen, as it is one of the major contributory factors for sterility.

Summary and Conclusions

Histochemical study of endometrium for glycogen was carried out in 189 out of 250 cases of Primary sterility. Hypoplastic and early proliferative endometrium showed nil to little amount of glycogen in the glands and the stroma. Moderate to good amount of glycogen was seen in the stromal cells of 18.3%, 15.8% and 10.7% cases of late proliferative early secretory and late secretory endometrium respectively. Impaired distribution of glycogen specially of stroma cells may be responsible for the poor preparation of the endometrium for implantation of fertilized ovum resulting in primary sterility.

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