Ovarian reserve in infertile women with chronic pelvic inflammatory disease

ABSTRACT

Objective: To assess ovarian reserve among infertile women with chronic pelvic inflammatory disease (PID).

Materials and Methods: A prospective study comprised of 35 women (study group A) with clinically and laparoscopically diagnosed PID and 15 cases as control. All cases were assayed for day 3 serum FSH, E2 and inhibin B.

Results: In group A, day 3 FSH & E2 were significantly higher than control (11.2±6.6 mIU/ml and 68.5±21 pg/ml versus 5.3±3 mIU/ml and 41.2±16 pg/ml, P < 0.05). While serum inhibin B was significantly reduced in group A (40±19 pg/ml) compared to the control (60±10 pg/ml). Serum inhibin B was negatively correlated with serum FSH in patients with PID.

Conclusion: Ovarian reserve appears to be relatively diminished in women with PID. This observation denotes progressive loss of ovarian reserve in cases of PID due to poor follicular development.

Key words: Ovarian reserve, Infertility, Chronic pelvic inflammatory disease.

INTRODUCTION

Ovarian reserve means the presence of sufficient number of follicles available for recruitment and development that will yield a cohort of eggs capable to lead successfully a conception cycle 1. Screening of ovarian reserve has been studied by many fertility centers to evaluate women reproductive potentials, both in the general fertility population 2, and for couples undergoing in vitro fertilization "IVF" 3.

PID is a common benign gynaecological disease that affects about 30% of infertile women based on clinical and laparoscopic evaluation. The association between infertility and PID is well established 4. The development of in vitro fertilization-embryo transfer (IVF-ET) has provided a new therapeutic approach for infertility. However, the results of IVF for patients with chronic PID are controversial. Several investigators had reported that the outcome of IVF was poorer for patients with PID if associated with poor ovarian reserve 5, 6.

Association between PID and poor follicular development has been proposed, resulting in abnormal steroid hormone production. So, ovarian reserve assessment is essential to identify the poor responder before initiation of controlled ovarian hyperstimulation or assisted reproductive programs, to lower the risk of cancellation and improve the pregnancy rates 7. The aim of the present study was to determine ovarian reserve in infertile women with PID and compare that to normal fertile women as a control.

MATERIAL & METHODS

This study was carried out in Departments of Obstetrics & Gynecology and Clinical Pathology, Faculty of Medicine, Mansoura University from the period of May 2007 to October 2009. Thirty five 35 patients with clinically and laparoscopically diagnosed PID (pelvic adhesion, pre-tubal adhesion, hydrosalpinx and pyosalpinx, in cases of exacerbation, edema, hyperemia and dilated blood vessels of the pelvic structures) 4 as study group. Fifteen women with normal reproductive outcome were as a control group, ultra-sonographic assessment was done to exclude any gross pelvic lesions. An informed consent was taken from all cases in the study.

At day 3 of menstruation, 3 blood samples were withdrawn from both patients and controls (2 ml) each at 10 minutes interval to avoid fluctuation into plain tubes. An equal volumes of the separated sera were pooled and kept frozen (-20°C) till analysis of serum FSH by chemiluminescence immunoassay using (immulite analyzer Dpc Los Angles) according to Babson 8, serum estradiol (E2) by electrochemiluminescence immunoassay using (Roche Elecsys 1010 immunoassay analyzer) according to method of Jonsen et al. 9 and serum inhibin B was assayed by enzyme immunoassay Kit Biosource-Belgium according to Groom et al. 10.
RESULTS

Table I show clinical data of study and control groups as regard age, BMI (body mass index) and cycle length. Table (II) show statistical data of hormonal assay comparison between study group A and control group. Table (III) for correlation coefficient between inhibit B and FSH & E2 in PID patients and control group confirmed the previous findings.

Table I: Clinical data of study group (55 cases) and control group (55 cases)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group</th>
<th>Study Group (n=55)</th>
<th>Control Group (n=55)</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>25 ± 3.6</td>
<td>23 ± 3.3</td>
<td>26 ± 2.9</td>
<td>-0.01</td>
<td>0.69</td>
</tr>
<tr>
<td>Body mass index (BMI)</td>
<td>28.6 ± 5.1</td>
<td>29.1 ± 4.8</td>
<td>28.5 ± 5.2</td>
<td>-0.01</td>
<td>0.72</td>
</tr>
<tr>
<td>Cycle length (d)</td>
<td>29 ± 4</td>
<td>28 ± 5</td>
<td>29 ± 6</td>
<td>-0.30</td>
<td>0.03</td>
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</tbody>
</table>

Table II: Hormonal assay comparison between study group and control group

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Study group (n=30 Mean±SD)</th>
<th>Control group (n=30 Mean±SD)</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FSH (mIU/ml)</td>
<td>11.2 ± 8.4</td>
<td>9.3 ± 5.3</td>
<td>-0.31</td>
<td>0.01</td>
</tr>
<tr>
<td>E2 (pg/ml)</td>
<td>69.5 ± 21.1</td>
<td>50 ± 17</td>
<td>-0.01</td>
<td>0.32</td>
</tr>
<tr>
<td>Inhibit B (pg/ml)</td>
<td>40.16</td>
<td>86.9 ± 20</td>
<td>-0.01</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Table III: Correlation coefficient between inhibit B and FSH & E2 in PID patients and control group

Inhibit B is one of the ovarian peptide (a heterodimeric glycoprotein) released by granulosa cells of the follicle early in the menstrual cycle having an inhibitory effect on the pituitary FSH release 8. So, it is considered as a direct marker of ovarian function. Low inhibit level was associated with poor response to superovulation. Measurement of basal inhibit B level serves as an attractive indicator of ovarian function as it probably precedes the increase in FSH precipitating its release 8.

Serum inhibit B was significantly reduced in group B PID (40 ± 18 pg/ml) compared to control (60 ± 10 pg/ml) but did not differ in group A compared to control. Inhibit B was negatively correlated with FSH level in patient with advanced PID in group B (Table 3). Defective follicular growth in PID results in reduction of inhibit B and defective steroidogenic activity. As the natural cycles in women with PID have been shown to have longer follicular phase 8. Diminished ovarian reserve in PID may appear quite logic. This may be attributed to the local destructive process that may be associated with chronic PID and/or fibrosis associated with the infertile lesion. Added to this explanation PID associated with the chemical and cellular local ovarian and pelvic milieu related to increased number of prostaglandins, activated macrophages and oxygen free radicals resulting in decreased inhibit B levels leading to increasing FSH which in turn increasing basal E2 17, 18, 19.

In conclusion, women with chronic PID have relatively diminished ovarian reserve. So, evaluation of patients with PID for ovarian reserve via serum inhibit B, FSH and E2 is essential before initiation of infertility treatment.

REFERENCES