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Hormonal Changes after Laparoscopic Ovarian Endocoagulation in Polycystic Ovarian Syndrome

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Abstract: **Objective is to study the effects of ovarian endocoagulation on the hormonal profiles in PCOS patients.**
Design: Prospective study.
Patients: 40 infertile patients, failed to respond to medical induction of ovulation.
Setting: Benha Teaching Hospital - laparoscopic unit of gynaecological department, Benha, Egypt.
Intervention: Laparoscopic ovarian endocoagulation.
Main outcome measure(s): Depending upon the size of the ovary, 4 – 10 holes to a depth up to 0.5cm were done in each ovary. Measurement of serum levels of LH, FSH, oestradiol, androstenedione, testosterone "total and free", prolactin and insulin was done one day before and one day, 3-7 days and about 2 months after ovarian endocoagulation.
Results: There were significant reductions in the levels of LH, oestradiol and androstenedione. Insulin level showed significant increases. There were no significant changes in serum levels of FSH and testosterones (free or total). Oestrogen level and ASD were positively correlated significantly with the ovarian cyst number. The ovarian volume and the number of ovarian microcysts were directly correlated significantly with ASD, free (T), and oestradiol.
Conclusion: Ovarian endocoagulation is an effective method in the management of anovulation in PCOS patients. It makes the hormonal environment - local in the ovary and systemic - suitable for ovulation. ASD is a reliable indicator in assessing the androgen status of the PCO patients. Increment of insulin level after endocoagulation may not support the role of insulin in the pathogenesis of PCOS.

Keywords: **laparoscopic ovarian drilling; ovarian diathermy; ovarian electrocautery; ovarian endocoagulation in PCOS.**

1. INTRODUCTION

Polycystic ovaries (PCO) including the polycystic ovarian syndrome (PCOS) are the most common endocrinopathy in women of reproductive age. PCOS remains a syndrome and no single diagnostic criterion (such as hyperandrogenism or polycystic ovaries) is sufficient for clinical diagnosis. PCOS is the commonest cause of anovulatory infertility; approximately 75% of anovulatory women have polycystic ovaries **(Farquhar et al., 2012)**.

Laparoscopic ovarian drilling (LOD), introduced by **Gjönnaess, 1984,** is an alternative to ovulation induction for patients unresponsive to clomiphene or gonadotrophins. Several laparoscopic approaches for ovarian drilling have been studied, and the results in the literatures are variable. LOD avoids the risks of multiple pregnancy and ovarian hyperstimulation

syndrome and enables full tubo-peritoneal assessment at the same time. However, there are ongoing concerns about adhesion formation and long-term effects on ovarian function **(**Flyckt and Goldberg **, 2011** and **Farquhar, et al., 2012).**

Of the many laparoscopic methods, no single technique has yet been found to be superior to the other. **Zdanovsky and his group, 1989** have used endocoagulation with the advantages of controlled destruction of the ovarian tissue and good pregnancy rates.

In our research, we have used the endocoagulator for ovarian drilling which permits the application of destructive heat without any risk of electric current within the abdominal cavity, and to our knowledge this is the second research whorled wide using endocoagulator.

2. PATIENTS AND METHODS

2.1. Patients:

This study was done in Benha Teaching Hospital, Gynaecology and Laparoscopy units. Forty PCOS patients were selected. The mean age was ± 26 years ± 4.24 SD. Most of the patients (72.5%) their ages were between 20 and 30 years

All the patients were seen because of infertility, and had spontaneous onset of puberty and normal sexual development. The mean duration of infertility was 6.3 years ± 4.2 SD. All the patients were anovulatory; clomiphene-resistant, and failed to respond to gonadotrophin injections. The diagnosis of PCOS was based on the following:

- The clinical diagnosis of PCOS with the presence of at least two of the following criteria: oligo-amenorrhoea, obesity (BMI ≥ 28), and clinical signs of hyperandrogenism or elevated either LH or LH/FSH ratio.
- Ultrasound evidence of polycystic ovary with the presence of ≥ 5 microcysts distributed peripherally.
- Laparoscopic demonstration of PCO with smooth, glistening white, thickened capsule with no evidence of ovulation.

Ovarian endocoagulation system used to heat the tissues of the ovary at 100°c, at which the protein coagulates.

In brief, laparoscopy was done through three-puncture technique using Wisap equipment; the laparoscope was introduced through the umbilicus and grasping forceps was introduced through a lower abdominal incision. After the shapes of the ovaries were confirmed to be polycystic with thick glistening capsule, tubal patency was assessed with methylene blue hydrotubation. The ovaries were fixed in such a position that damage to other organs was avoided. Through a third puncture site, the pointed SEMM endocoagulator instrument (WISAP/ FRG) **(Picture: 1)** was held against the surface of the ovary for 2 to 4 seconds until it is seen to penetrate the cortex. This was accompanied by a popping sensation and was often associated with a burst of fluid that exits from subcortical follicles.

Picture (1): Endocoagulator instrument (WISAP/ FRG)

The number of holes for each ovary was usually "between" 4-7, depending upon the ovarian size. Each hole is approximately 3-5 mm in diameter and 2-5 mm in depth. They were not too close to the fimbrial pole, to avoid adhesions in this area. No drilling was done within 8 to 10 mm of the mesovar. Hemostasis was achieved, if necessary, with additional use of endocoagulation. After irrigation and cooling of the ovary, artificial ascites ± 500 ml of sterile Ringer Lactate usually left after completion of the procedure.

After ovarian endocoagulation, the women were followed up regularly for 3months.

2.2. Ultrasound Examination:

All women had undergone transvaginal ultrasound, using a 5 MHz transducer (Combison ^{R} 310 a Kretztechnik ultrasound machine). An ultrasound was done before operation in the early follicular phase between the 2nd and 8th day of the menstrual cycle, and between 3rd and 7th day, 2nd and 3rd week postoperative and then routine ultrasound screening for

ovulation in the first three months following the operation (without medication). Ovulation was documented if the dominant Graffian follicle exceeded 18 mm in diameter with either consequent disappearance or intrafollicular bleeding in the following few days usually with the existence of fluid in the Douglas pouch. In amenorrhoeic women, the investigations were performed after progestogen induced withdrawal bleeding.

The ovary was defined as being polycystic if there were multiple (more than four), small (2-8 mm) cysts arranged peripherally. In all cases, both ovaries were visualized. Assessment of the stromal echodensity (hypertrophy), due to its subjectivity, was not included in the criteria for polycystic ovaries. The ovary was measured in three planes, then the ovarian volume was calculated according to the formula for a prolate ellipsoid: (d1) x (d2) x (d3) x 0.5233, where the d1, d2, d3 are the three maximal longitudinal, anteroposterior and transverse diameters. Ovaries were considered enlarged when the ovarian volume exceeded 9 ml. Ovaries containing cysts (a dominant follicle, simple cyst, haemorrhagic cyst, endometrioma, and corpus luteum) > 14 mm in diameter were excluded. The patients were divided into 3 groups; group (1), of less than 10 cysts; group (2), between 10 and 14 cysts; group (3), of more than 14 cysts.

2.3. Hormonal Measurements:

All blood samples were usually obtained on the same day that US were performed. The blood was taken (fasting) one day before operation, one day, three to seven days, and about two months (cycle day two – three of the menses) after ovarian endocoagulation. In each time, the following hormones were assayed: LH, FSH, oestradiol, ASD, total (T), free (T), insulin and prolactin.

2.4. Statistics:

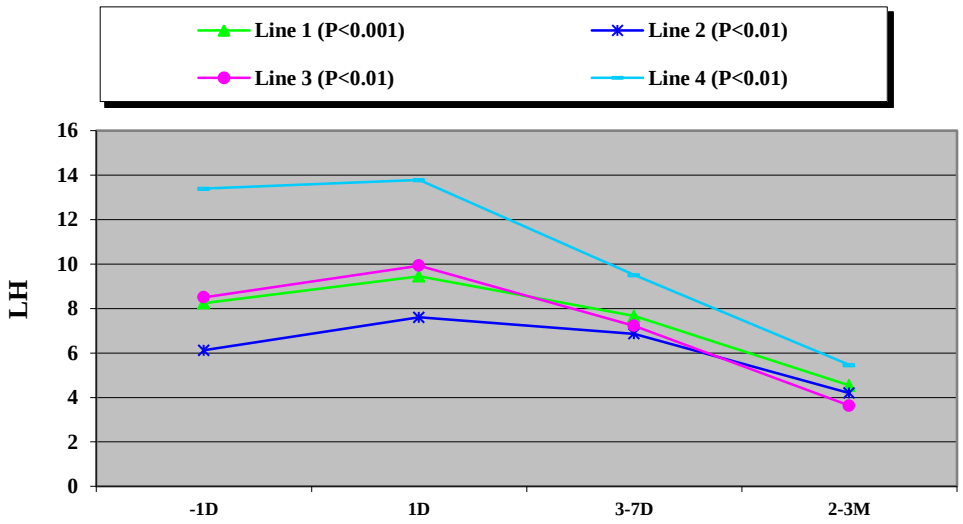
Analysis of data was performed using the Statistical Package for Social Sciences computer program (SPSS/PC+). The data were analyzed using the analysis of variance (ANOVA). Pearson's product-moment coefficient *r* was also used to test the correlation between the endocrine and the biophysical data after log transformation. Analysis of variance after log transformation and chi-square was used in addition to T-Test and Z-Test, when appropriate, to test the differences between groups.

3. RESULTS

In order to analyze the treatment data, the selected PCOS patients were classified into five groups: according to the preoperative serum LH levels (> 11 mIU/ml and ≤ 11 mIU/ml), ovarian volume (> 9ml and ≤ 9 ml), in addition to all patients altogether.

3.1. Endocrinological Findings:

Figure (1):The effect of ovarian endocoagulation on serum LH levels (mIU/ml) in classified PCOS patients:



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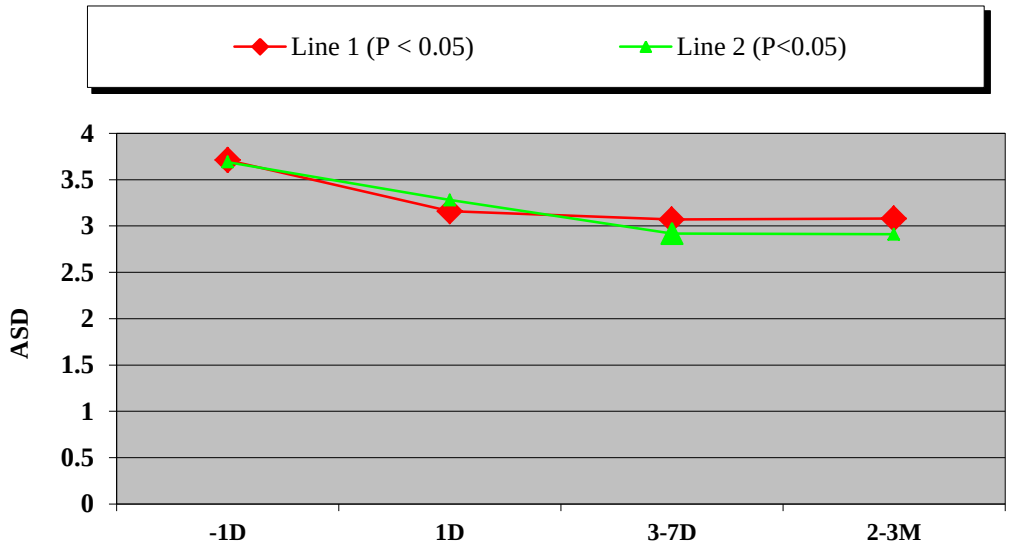
N.B.: **-1D** = one day before endocoagulation; **1D** = one day after endocoagulation; **3-7D** = 3-7 days after endocoagulation; **2-3M** = 2-3 months after endocoagulation. **Line 1**= all patients; **Line 2**= patients with LH ≤ 11mIU/ml; **Line 3**= patients with ovarian volume ≤ 9ml; **Line 4**= patients with LH > 11mIU/ml.

Raised preoperative serum LH in the early follicular phase (>11 mIU/ml) was found in 12 patients (12/40) (30%). LH/FSH ratio ≥ 2 was found in 16 patients (40%) and ≥ 2.5 in 7 patients (17.5%). **In figure (1):** there was significant reduction in LH secretions in most of the classified PCOS patients. There are initial rises in serum LH levels on day one after operation, followed by gradual decline to reach the nadir, below the pretreatment level, about two months after operation. There were no significant changes in serum LH in patients with ovarian volume > 9 ml.

There was no significant change in the serum levels of FSH. However, the hormone was elevated on day one after the operation, especially in those patients that showed significant changes in estradiol levels.

Figure (2):The effect of ovarian endocoagulation on serum androstenedione (ASD) levels (pg/ml) in classified PCOS patients:



N.B.: **-1D** = one day before endocoagulation; **1D** = one day after endocoagulation; **3-7D** = 3-7 days after endocoagulation; **2-3M** = 2-3 months after endocoagulation. **Line 1**= patients with LH levels ≤ 11 mIU/ml; **Line 2**= patients with ovarian volume > 9ml.

In figure (2): androstenedione levels were significantly decreased in those with preoperative LH levels ≤ 11 mIU/ml (P <0.05) or LH ≤ 10 mIU/ml (P < 0.01) and with ovarian volume > 9 ml. The levels of androstenedione decline gradually starting from the first day postoperative and reach its lowest level nearly two months after operation.

Testosterone levels, total or free, did not show any significant changes. However, the levels after the operation were apparently less than the preoperative ones.

Raised preoperative serum androstenedione in the early follicular phase (>2.7 ng/ml) was found in 28 of the selected PCOS patients (28/39) (71.8 %). Serum total testosterone levels > 1.0 ng/ml was found in 16 patients (16/38) (42.1%), while serum free testosterone > 3.2 pg/ml was found in 21 patients (21/40) (52.5%). Of 22 patients with normal total testosterone levels, 14 patients (63.6%) had an elevated androstenedione. Whereas two patients out of 11 with normal androstenedione levels (18.2%) had elevated total testosterone. Of those 16 patients with elevated total testosterone, only one patient had normal androstenedione level. On the other hand, of 19 patients with normal free testosterone, eight patients had elevated androstenedione levels. Whereas only one patient out of 21 with elevated free testosterone had normal androstenedione level, and only one patient out of 11 with normal androstenedione had elevated free testosterone.

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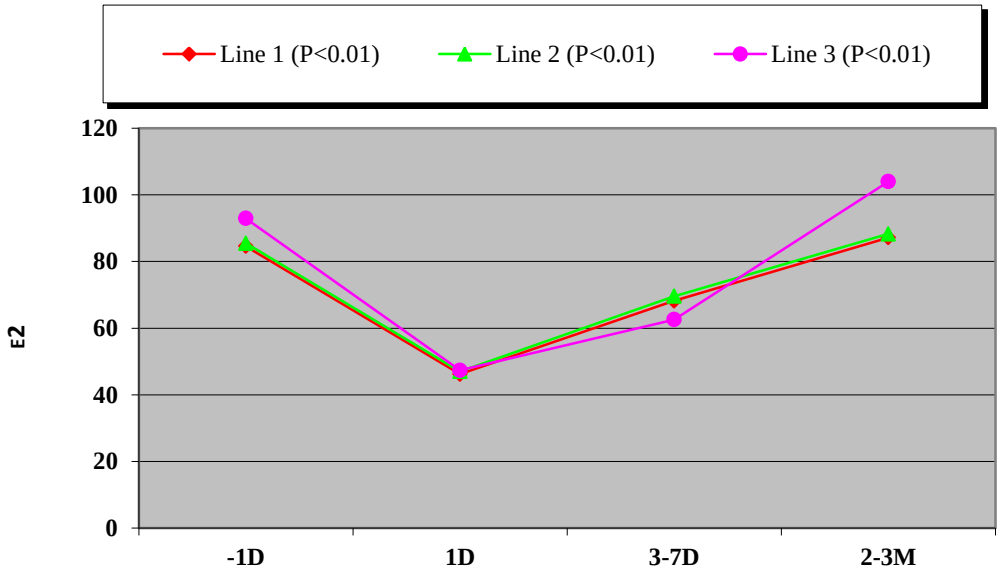
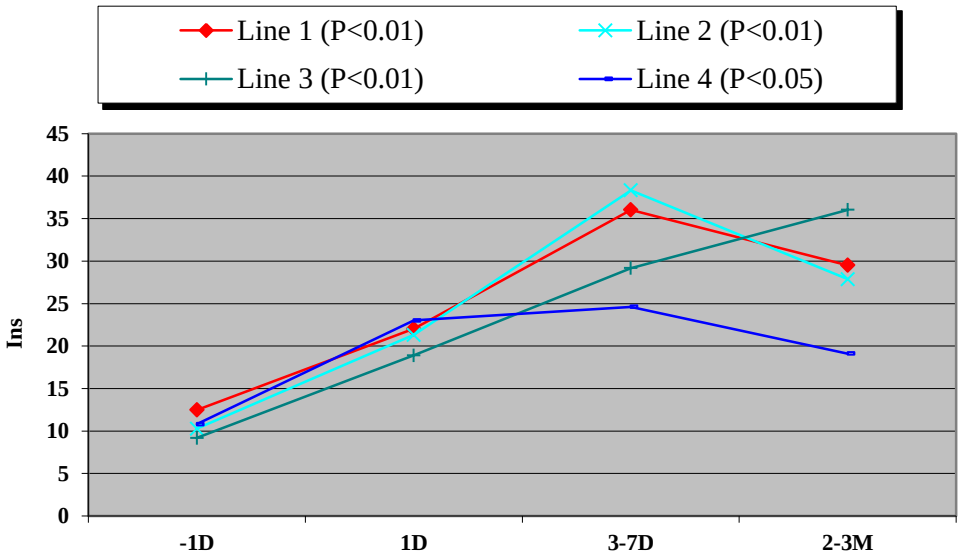
Figure (3):The effect of ovarian endocoagulation on serum oestradiol levels (pg/ml) in classified PCOS patients:



N.B.: **-1D** = one day before endocoagulation; **1D** = one day after endocoagulation; **3-7D**= 3-7 days after endocoagulation; **2-3M**= 2-3 months after endocoagulation. **Line 1**= all patients; **Line 2**= patients with LH ≤ 11mIU/ml; **Line 3**= patients with ovarian volume >9ml.

In figure (3): the oestradiol levels decline and reach the nadir one day after ovarian endocoagulation and then start to rise again to reach the pretreatment levels, or slightly above, about two months after operation. There was no significant change in oestradiol levels in those groups of patients with LH > 11 mIU/ml and ovarian volume ≤ 9 ml.

Figure (4):The effect of ovarian endocoagulation on serum insulin levels (micIU/ml) in classified PCOS patients:



N.B.: **-1D**= one day before endocoagulation; **1D**= one day after endocoagulation; **3-7D**= 3-7 days after endocoagulation; **2-3M**= 2-3 months after endocoagulation. **Line 1**= all patients; **Line 2**= patients with LH ≤ 11mIU; **Line 3**= patients with ovarian volume ≤ 9ml; **Line 4**= patients with ovarian volume > 9 ml.

In figure (4): serum insulin levels rise steadily from day one to day seven and then gradually declines however, the levels remained higher than the pretreatment levels.

The incidence of hyperprolactinemia in our patients was 20% (8/40). The prolactin concentration in those hyperprolactinemic patients was 780.36 ± 205.4 µIU/ml (mean ± SD). Only in all patients altogether group, serum prolactin levels showed significant changes ($P < 0.05$); in which the serum prolactin levels significantly decreased to the nadir in the first week after ovarian endocoagulation and after about two months, its level was nearly as the pretreatment level.

3.2. Ultrasound Findings:

In the selected PCOS patients, most of the ovaries in the right side (70%) and the left side (72.5%) were > 9 ml. 40-42.5% of the patients had between 10-14 microcysts in each ovary. Average values from both ovaries were used for further analysis. Most of the microcysts (87.5% from right ovary and 92.5% from left ovary) were distributed subcuticularly.

In **table (1):** only ASD levels were significantly ($P < 0.05$) higher in group (III) and (II) than in group (I) of the ovarian cysts. In addition, it was significantly increased ($P < 0.05$) in patients with ovarian volume > 9 ml than in patients with ovarian volume ≤ 9 ml.

Table (1): Hormonal changes before and after ovarian drilling depending on the ovarian ultrasound findings:

Variable	Ovarian cysts			Ovarian volume	
	Gr.I **<10 cysts** (n=12)	**Gr.II** **10-14 cystsb** (n=16)	**Gr.III** **>14 cysts** (n=12)	**≤ 9ml** (n=14)	**> 9ml** (n=26)
LH –1D	**8.81*** **	**7.42**	**8.77**	**8.76 ****	**7.96**
1D	**10.38**	**9.0**	**9.12**	**9.95**	**9.18**
3-7D	**7.46**	**8.14**	**7.37**	**7.8**	**7.56**
2-3M	**3.46**	**4.55**	**5.72**	**3.6**	**5.08**
FSH –1D	5.54	4.54	5.08	5.77	4.59
1D	5.63	5.0	5.06	5.33	5.14
3-7D	5.38	4.59	4.7	5.29	4.63
2-3M	5.12	4.29	4.27	4.83	4.38
Oestradiol –1D	**61.39**	**74.71 ****	**122.2 ***	**70.29**	**92.95 ****
1D	**47.78**	**4 6.54**	**41.3**	**44.31**	**47.49**
3-7D	**83.63**	**51.19**	**68.07**	**75.7**	**62.55**
2-3M	**51.16**	**106.7**	**107.74**	**64.81**	**103.99**
Total testosterone –1D	0.89	1.18	1.12	**0.91**	**1.17**
1D	0.88	0.99	0.9	**0.86**	**0.97**
3-7D	1.03	0.81	0.87	**1.0**	**0.84**
2-3M	0.87	0.97	1.09	**0.85**	**1.06**
Free testosterone –1D	**2.94**	**4.0**	**4.28**	**3.59**	**3.86**
1D	2.88	3.19	3.36	**2.84**	**3.31**
3-7D	3.28	3.42	2.48	**3.49**	**2.72**
2-3M	2.79	3.63	3.46	**2.69**	**3.64**
Androstenedione -1D	**2.73**	**3.58**	**3.88 (+)**	**2.96**	**3.69 * (+)**
1D	2.75	3.29	3.28	**2.86**	**3.28**
3-7D	2.49	3.03	3.5	**3.03**	**2.9**
2-3M	2.5	2.89	3.2	**2.73**	**2.91**
Prolactin -1D	246.6	352.3	407.4	260.7	378.2
1D	345.1	310.0	304.4	335.1	310.1
3-7D	196.1	206.2	227.7	201.0	216.3
2-3M	321.9	520.6	259.1	260.6	428.1
Insulin -1D	**9.67**	**12.72**	15.2 * **6.98 (c)**	**9.18 ****	14.33 **10.8 (b)***
1D	25.12	26.01	13.68	**18.91**	23.73 **23.01**
3-7D	17.31	36.96	55.72 (a)	**29.18**	40.61 **24.53**
2-3M	25.71	39.58	21.34	**36.03**	25.05 **19.11**

All values are mean. **-1D**= one day before endocoagulation; **1D**= one day after endocoagulation; **3-7D**= 3-7 days after endocoagulation; **2-3M**= 2-3 months after endocoagulation; **(a)**= two patients their insulin levels were > 126 uIU/ml. **(b)**= After excluding four patients; one from insulin-**1D** (insulin level > 97 µIU/ml) and two from insulin **3-7D** and one from insulin **2-3M** (insulin levels were >126 µIU/ml). **(c)** After excluding one patient her serum insulin (**-1D**) was >97 µIU/ml. **(+)** $P < 0.05$ comparing values between groups. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ comparing values before and after drilling.

The spontaneous ovulation rate following endocoagulation was 54.3% (19 /35 patients). Ovulation occurred between the16th and the 32-day after endocoagulation.

3.3. Correlations:

The number of ovarian microcysts was directly correlated significantly with the serum levels of androstenedione ($r = 0.401$, $P = 0.000$), oestradiol ($r = 0.233$, $P = 0.007$) and with free testosterone ($r = 0.199$, $P = 0.022$) but not with total testosterone ($r = 0.162$, $P = 0.067$). Ovarian volume was significantly correlated with androstenedione ($r = 0.282$, $P = 0.002$), total and free testosterone ($r = 0.327$ and $r = 0.356$, respectively, $P = 0.000$), and oestradiol ($r = 0.181$, $P = 0.039$). On the other hand, ovarian volume was not correlated with serum levels of LH ($r = -.103$, $P=0.25$), FSH ($r = -0.171$, $P = 0.06$), prolactin ($r = 0.031$, $P = 0.74$), insulin ($r = 0.146$, $P = 0.122$) or the duration of infertility ($r = -0.158$, $P = 0.07$). The ovarian volume was negatively correlated significantly ($r = -0.265$, $P = 0.002$) with the patients' age.

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4. DISCUSSION

The mechanism of LOD is still unclear. Direct damage to both ovarian capsule and stroma may result in release of local cascades or decrease levels of ovarian steroids and non-steroids. Consequently, this will change pituitary sensitivity and secretion. Normalization of ovarian cycles may be linked to a factor (s) found on the capsular surface of the ovary, rather than in the stroma **(Campo et al., 1983)**. Postoperative ovarian inflammation may increase the delivery of gonadotropins to the ovary **(Kovacs et al., 1991).** In addition, the effect of anaesthesia and the operation context may be responsible for the immediate hormonal changes **(Hendriks et al., 2014)**.

In our study, the LH finding was coinciding with other researches; after laparoscopic ovarian electrocautery (**Farhi et al., 1995)**, after endocoagulation **(Zdanovsky et al., 1989)** and after laser surgery **(Hendriks et al., 2014).** However, there are other studies with no initial rise in the level of LH after using electrocautery **(Demirturk et al., 2006)**, after thermo aqua puncture **(Ramzy and Ramzy, 1999)** and after laser **(Takeuchi et al., 2002)**. Moreover, there are reports with no changes in LH, after electrocautery **(Kovacs et al., 1991)** and after laser **(Asada et al., 2002). Armar et al. (1990)** Found LH values variable after ovarian electrocautery.

We did not find any significant change in the serum levels of FSH, this was similar to other results obtained after laser vaporization **(Verhelst et al., 1993**) and after electrocautery **(**Parsanezhad ***et al.,*** 2005**).** However, significant increase had been reported after laser (**Hendriks et al., 2014)**, after thermo aqua puncture **(Ramzy and Ramzy (1999)** and after electrocautery **(Demirturk et al., 2006)**. Conversely, others **(Alborzi et al., 2001)** have found marked decrease in the level after ovarian electrocautery.

The lowest level of oestrogen in day one postoperative was corresponding to the highest level of both FSH and LH. The changes in estradiol levels in our studies may be explained by the destruction of oestrogen secreting cells inside the ovary in addition to the decreased androgen conversion to oestrogen. Our results were similar to results obtained after

electrocautery (**Kovacs et al., 1991)**, and laser surgery **(Rossmanith et al., 1991)**. In another study **(Greenblatt and Casper, 1987)** after using ovarian cautery, the nadir was on day four postoperative. However, after laser vaporization **(Hendriks et al., 2014)** and cautery **(Kucuk and Okman, 2005)**, the estradiol level was slightly increased or showed no change, respectively.

In all patients altogether group, as in the research of **Alborazi (1994)**, the level of serum prolactin has significantly decreased in the first week following ovarian drilling then started to rise again. The explanation is unclear. In other studies, the level was increasing (**Parsanezhad et al., 2005)**, or showed no significant change (**Kucuk and Okman (2005)**.

Serum insulin levels after endocoagulation were significantly higher than the pretreatment levels. This is in accordance with the finding of **Abdel Gadir et al. (1990)**, but in other reports, the level was decreasing (**Api et al. (2005)** or remained unchanged **(**Malkawi et al, 2003**)**.

In our study, neither LH nor insulin had direct correlation with the androgens levels. This may not support the direct role of LH or insulin to cause PCOS or may denote that the specific local effect of any hormone may not be reflected by the peripheral serum concentrations.

In those patients with preoperative LH > 11mIU/ml, the only significant change after ovarian drilling was on the LH levels. Those patients with LH ≤ 11 mIU/ml, plus to the significant effect on LH levels, they showed significant effects on ASD, oestradiol, and insulin levels. This may demonstrate the role of LH in the genesis and maintenance of anovulation, and in the meantime and coinciding with the opinions of **Filicori et al. (1994),** may denote the importance and the possible synergistic role of insulin in the ovulatory process.

Depending on the number of the ovarian microcysts, group I (< 10 microcysts), group II (10-14 microcysts) and group III (> 14 microcysts), and coinciding with the finding of **Takahashi et al., 1994**, the correlation between these groups**,** was only significant in ASD levels. The numbers of ovarian microcysts were directly correlated significantly with the ovarian volume **(**coinciding with the finding of **Takahashi et al., 1995)**, and with ASD (coinciding with the finding of **Sikka et al., 2007)**, and with free (T) (coinciding with **Yoshino et.al. 1993)** and estradiol but not with total (T), LH, insulin or prolactin. In addition, the ovarian volume was significantly correlated with total and free (T) and with ASD (coinciding with the findings of (**Sikka et al., 2007**), and with oestradiol, but there was no correlation (as also reported by **El Tabbakh et al., 1986)** with serum levels of LH, FSH, prolactin. Consequently, we may assume the primary role of ovary in androgen production, and at the same time the significant predictive value of ovarian cyst number and ovarian volume for the androgen levels **(Sikka et al., 2007)**. In our research, only elevated ASD was observed in PCO patients with normal size ovary and in PCO patients with microcysts number <10 in one ovary. This may reflect the reliability of ASD in assessing the androgen status of PCO patients; this is in accordance with the observations of **Abdel Gadir et al. (1990)** and **Takahashi et al. (1993)**. Therefore, we may consider ASD combined with the Free (T) as a reliable marker for the diagnosis of PCOS**.**

5. CONCLUSION

We may recommend ovarian endocoagulation for management of anovulation in PCOS patients after exhaustion of the available medical methods for ovulation induction. Endocoagulation is a comparable method to the other types of ovarian drilling; it is a relatively safe as there is no electric current and the adhesion formation is negligible. Endocoagulation significantly decreases LH, ASD, estradiol levels. In addition, it significantly increases insulin level. ASD is a reliable indicator in assessing the androgen status of PCO patients.

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