Laparoscopic Ovarian drilling as Treatment for PCO Syndrome

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Diploma of obstetrics and gynecology-cairo university

For partial fulfillment of master degree in

Obstetrics and Gynecology

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2010

ACKNOWLEDGEMENT

I am immensely and deeply grateful to **prof. Dr Mostafa Mahmoud Asem**, Professor of Obstetrics and Gynecology , cairo university , for without his untiring guidance and faithful efforts , this work would never have been completed.

I am greatly indebeted and much grateful to **prof. dr. Mohamed Hany Shehata ,** Professor of Obstetrics and Gynecology , cairo university , for her huge assistance and sincere help.

I would like to profound gratitude to **Dr. Ahmed M.Mostafa El-Halwagy** Assistant Professor of Obstetrics and Gynecology cairo university , for his guidance , supervision and continuous noble-minded encouragement.

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Abstract

Laparoscopy is the accepted treatment modality for many gynecological conditions. Offering patients a shorter hospital stay. faster recovery. Less need for analgesia and a better cosmetic appearance. Many complications specific to laparoscopy. Can cause major morbidity and potential mortality(Jacobson et ai, 2004).

Complication of laparoscopy include, de novo Adhesion Formation Inadvertent Extraperitional Insufflatiocardiovascular Complications Gas Embolismvascular Injuries Gastrointestinal Injuries Urinary Tract Injuries Electrosurgical complications Ovarian failure.

Key words:

Laparoscopic Ovarian drilling

Treatment for PCO Syndrome

Content:

Introduction:

Polycystic Ovary Syndrome (PCOS.) is a common endocrine disorder in women of reproductive age with primary manifestations of infertility. menstrual dysfunction, and clinical or biochemical hyperandrogenism (hirsutism, acne, and elevated androgens) (Moran et al 2003).

Polycystic ovary syndrome, one of the most common endocrine disorders, affects approximately 6 percent of women of reproductive age. The syndrome is the most frequent cause of anovulatory infertility, with its underlying etiology unknown. The classic description . of the syndrome which includes clinical findings of amenorrhea hirsutism and bilaterally enlarged ovaries, is representative of more advanced cases (Bachmann, 1998).

Polycystic ovary syndrome is now recognized as a heterogeneous Syndrome. Affected women often have signs and symptoms of elevated androgen levels. menstrual irregularity. and amenorrhea without a well-defined cause of androgen excess. The syndrome has an initial onset in the peripubertal years and is progressive (Hunter .and Pharm, 2000).

Recent successes with ovulation-inducing agents have decreased the use of ovarian wedge resection surgery. Newer surgical techniques such as ovarian drilling often provide temporary results and do not address the underlying metabolic .disturbances in patents with polycystic ovary syndrome. A significant percentage of Women who undergo ovarian cautery via laparoscopic techniques

have spontaneous restoration of ovulation with subsequent pregnancy, but postoperative complication , including adhesion formation tend to overshadow the potential benefits of these surgical interventions(Goudas and Dumesic, 1997, Hunter and Pharm, 2000)

In the 1920 When PCOS first recognized , there was only one form of treatment that was known to be effective in initiating ovulation ovarian wedge resection . in the years then the practice fell out of favor in the medical community due to the risk of surgery itself and of postsurgical adhesion formation . in 1984 laparoscopic diathermy or ovarian drilling were introduced as anew modality for treatment of PCOS (Pritts ,2002)

Surgical treatment of PCOS was first introduced almost one century ago when ovarian wedge resection by laparotomy was performed with good results . This procedure resulted in spontaneous ovulation and become popular in the 1950s. A significant reduction in androgen levels was observed. Resulting in normal ovulation. Unfortunately the effect was short-lived and was associated with an increased likelihood of periadnexal adhesion. Among woman who failed to conceive , a diagnostic laparoscopy demonstrated pelvic adhesion in 15-100%. As a result , ovarian wedge resection fell into disrepute and was largely abandoned.(daya,2001)

Recently interest in surgical treatment of PCOS has been renewed with the increased availability and use of operative laparoscopy. Several techniques have been introduced to restore ovulation in women with clomiphene citrate-resistant PCOS . these

techniques includes ovarian biopsy, multiple ovarian punch biopsy ovarian capsule resection , ovarian electrocautery , and laser vaporization . the rationale for the surgical approach is unchanged . i.e reduction of ovarian androgen levels so that follicular development can take place by avoiding the atresia that results from excess androgens (Gurgan et al 1994)

There is insufficient evidence amongst women with polycystic ovarian syndrome and clomiphene resistance that either laparoscopic ovarian drilling or ovulation induction with gonadotrophins is superior for the outcomes of pregnancy and ovulation (with the exception of multiple pregnancies which are almost nonexistent with ovarian drilling) (Farquhar et al 2002)

With regard to adhesion formation . there is insufficient evidence to favour any one surgical technique over another . therefore at present , until more data become available , the clinical decision as to which treatment to recommend could be made on other grounds such as local facilities side effects , cost and patient acceptability (Farquhar et al 2002)

In a study by(Ligouri e al 1996) , the incidence of de novo adhesions after laparoscopic ovarian cauterization was found to be 23.3, The evidence to date indicates that laparoscopic ovarian electocautry is a promising intervention to restore ovulation.

However. It is important to study de novo adhesion formation and its effect on fertility.

Aim of the work

Illustration of cons and pros of laparoscopic management of polycystic ovarian disease.

The definition of polycystic ovary syndrome has been reviewed: in 1935, Stein and Leventhal described a symptom complex that consisted of a clinical triad of hyper-androgenism, an-ovulation and obesity in women with enlarged polycystic ovaries; the current diagnostic criteria are hyper-androgenism and ovulatroy dysfunction with the exclusion of a well defined underlying cause of androgen excess. (Adriaanse., et al 1998)

Acceptance of this syndrome as a singular clinical entity led to a rather rigid approach to have a history of oligomenorrhea, hirsutism, and obesity, together with the demonstration of enlarged polycystic ovaries, a clinical state now recognized to be characteristic of extreme cases that ware included. It is better to consider this problem as one of persistent an-ovulation with a spectrum of aetiologies and clinical manifestations, after exclusion of specific conditions such as adrenal hyperplasia, Cushing syndrome, hyper-prolactinemia, and androgen producing tumours. (Speroff., al.,1999)

Polycystic ovarian syndrome (PCOS) is the commonest endocrine disorder in women of a reproductive age, occurring in approximately one in seven women. Of these women approximately two-thirds will not on a regular basis and consequently may therefore seek treatment for ovulation induction. After exclusion of other significant causes of sub-fertility the pragmatic approach to ovulation induction is to commence with clomiphene citrate. The goal of ovulation induction is the development of a single ovulatory follicle and the avoidance of a multiple gestation. Second line therapies consist of gonadotrophin therapy and laparoscopic ovarian drilling,

the place of metformin therapy is believed to lie in the management of woman with impaired glucose tolerance. The benefit of the use of aromatase inhibitors has not yet been proven in large studies. Women with PCOS undergoing in vitro fertilization (lVF) are at a substantial risk of ovarian hyperstimulation syndrome and this approach should be avoided if at all possible. If it is required these women may be suitable candidates for in vitro maturation of oocytes (lVM) so avoiding ovarian hyperstimulation. Women with PCOS are potentially at an increased risk of miscarriage and in pregnancy of they are at an increased risk of developing gestational diabetes, pregnancy-induced hypertension and pre-eclampsia. Furthermore the neonate has a significantly higher risk of admission to a neonatal intensive care unit and a higher perinatal mortality.(Hart R, 2008).

Prevalence of polycystic ovaries in women with reproductive failure:

Ultrasound evidence of polycystic ovaries is seen in 20-23% of apparently normal women, but the prevalence of polycystic ovaries is increased in women with reproductive failure. (Farquhar., et al 1994)

Women presenting with recurrent miscarriage have been shown to have a prevalence of polycystic ovaries ranging from 44-82%. In a large series of 500 consecutive women attending a specialized recurrent miscarriage clinic, prevalence of polycystic ovaries on ultrasound, scanning was 56% (Clifford., et al 1994)

It is argued that normally ovulating women with polycystic ovaries on ultrasonography have underlying metabolic abnormalities. However, the great majority of ovulatory women with polycystic ovaries on ultrasonography are endocrinologically normal, and only occasionally the androgen level mildly elevated. (Carmina., al 1997)

Etiology of polycystic ovarian disease:

I-Endocrinological Causes:

A-Hypothalamic and pituitary abnormality:

Normal pituitary ovulatory response to the follicle steroid signals requires the presence of gonadotrophin-re1easlng hormone pulsatile secretion within a critical range. A variety of problems, such as stress and anxiety, borderline anorexia nervosa, and acute weight loss after a crash diet, are associated with an inhibition of normal gonadotrophin-releasing hormone pulsatile secretion, due to excessive hypothalamic activity in response to stress. (Salinikis.,et al 1996) Hyper-prolactinemia has been recognized as one specific clinical syndrome of central an-ovulatory dysfunction. Hyper-prolactinemia can cause a woman to progress through a spectrum, beginning with an in adequate luteal phase to an-ovulation to the amenorrhoea associated with complete FSH suppression.

(Gnassi., et al 1996)

An-ovulatory women with polycystic ovaries have a higher LH pulse frequency and amplitude when compared the normal mid-follicular phase. The elevated Latinising hormone levels are partly due to increased sensitivity of the pituitary to releasing hormone stimulation manifested by an increase in Lutinising hormone pulse amplitude and frequency, but mainly amplitude. (Hayes., et al 1998)

B-Abnormal feedback Signals:

1- Loss of Follicle stimulating hormone stimulation:

A decrease in blood sex steroids is necessary to achieve recycling, by permitting the rise in Follicle stimulating hormone to take place. The necessary decline in blood oestrogen requires reduction of secretion, appropriate clearance and metabolism and the absence of a significant contribution of oestrogen to the circulation by persistent oestrogen secretion, encountered rarely with an adrenal or ovarian tumour. (Speroff., al.,1999)

2-Abnormal Oestrogen Clearance and Metabolism:

The clearance and metabolism of oestrogen can be impaired by other pathologic conditions such as thyroid or hepatic diseases. Both hyperthyroidism and hypothyroidism can cause persistent an-ovulation by altering not only metabolic clearance but also peripheral conversion rates among different steroids. (Speroff., al.,1999)

3-Extraglandular Oestrogen Production:

Although the adrenal gland does not secrete appreciable amounts of oestrogen into the circulation, it directly contributes to the total oestrogen level. This is accomplished by extra-gonadal peripheral conversion of C-19 androgenic precursors, mainly androstenedione, to oestrogen. In this way, psychological or physical stress may increase the adrenal contribution of oestrogen precursor, and subsequent conversion to oestrogen may sustain the blood level of oestrogen at a time when a decline is necessary for successful

recycling of the menstrual cycle. Adipose tissue is capable of converting androstenedione to oestrogen; hence the percent conversion increases with increasing body weight . (Plymate., et al 1981)

4-Loss of Lutinising hormone stimulation:

The failure to achieve a critical mid-cycle level of estradiol necessary to trigger the gonadotropin surge may be due to a relative deficiency in steroid production. This inadequacy may be due to intrinsic follicular weakness or impairment in the follicular gonadotrophin interaction. The end result is the same, which is failure to achieve critical signal levels of estradiol at the appropriate time in the mid-cycle. (Speroff., al.,1999)

5- Concentration of androgens in the ovarian follicle:

The androgens in low concentrations enhance aromatase activity and oestrogen production, by serving as substrate for FHS induced aromatisation. At higher concentrations, the granulosa cells favour the conversion of androgens to more Potent 5alpha reduced androgens, that cannot be converted to oestrogen and, in addition, are capable of inhibiting aromatase .activity and follicle stimulating hormone induction of lutinising hormone receptors. Thus, raising the local androgen concentration above' a critical level inhibits the emergence of a dominant follicle and leads to follicular atresia, prevent normal cycling and cause chronic an-ovulation . (Speroff., al.,1999)

II- Metabolic factors:

Obesity per se is associated with the development of insulin resistance at a post receptor level, and hyper-insulinemia subsequently results. Increased circulating insulin levels suppress both sex hormone binding globulin and insulin like growth factor I binding protein, thereby rendering a larger fraction of sex steroids and 'insulin like growth factor I biologically available. (Kaser .,1995)

2-Growth hormone and insulin like growth factor:

Normal puberty is characterized by the development of insulin resistance. The abnormality appears to be largely confined to peripheral not hepatic glucose metabolism. (Amiel., et al 1986)

The realization that normal puberty shares two key biochemical features with polycystic ovarian disease, namely increased adrenal androgen synthesis and insulin resistance in conjunction with the clinical impression that the disorder is usually noted to be of peri-pubertal onset, led naturally to the hypothesis that the development of polycystic ovarian disease is connected in some fundamental way to the normal developmental endocrine alterations that accompany puberty. (Kaser .,1995)