

**COMPARATIVE STUDY BETWEEN
LASER VAPORIZATION AND ELECTROCAUTERY
OF THE OVARIES IN POLYCYSTIC
OVARIAN DISEASE**

THESIS

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LIST OF ABBREVIATIONS

A	Androstenedione
ACTH	Adreno-cortico-tropic hormone
3 a - diol G	3 alpha-androstanediol glucuronide
C.C.	Clomiphene citrate
DHEAS	Dehydro-epiandrosterone sulfate
DHT	Dihydro-testosterone
E ₂	Estradiol
FAI	Free androgen index
FSH	Follicle-stimulating hormone
HCG	Human chorionic gonadotropin
HMG	Human menopausal gonadotropin
H.P.O. axis	Hypothalamo-pituitary-ovarian axis
IGF1	Insulin like growth factor -1
17 KSR	17 ketosteroid reductase
LASER	Light amplification by stimulated emission of radiation
LH	Leuteinizing hormone
LH-RH	Leuteinizing hormone releasing hormone
MASER	Microwave amplification by stimulated emission of radiation.
Nd : YAG	Neodymium-yttrium Aluminium Garnet
PCO	Polycystic ovaries
PCOD	Polycystic ovarian disease
PDT	Photo-dynamic therapy
SHBG	Sex hormone binding globulin
T	Testosterone
W	Watt

CONTENTS

	Page
INTRODUCTION	1
AIM OF THE WORK	4
REVIEW OF LITERATURE	
* Polycystic Ovarian Disease	5
* Laser Therapy in gynecology.....	140
PATEINTS AND METHODS	215
RESULTS.....	233
DISCUSSION	263
SUMMARY AND CONCLUSIONS.....	279
REFERENCES.....	284
ARABIC SUMMARY	----

INTRODUCTION

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Chereau first recorded the finding of sclerocystic change of the ovary in 1844.

It was the report by *Stein and Leventhal (1935)* of the association between absent or infrequent menstrual cycles, hirsutism, obesity and enlarged cystic ovaries that firmly established the association with ovarian dysfunction. (*Stein and Leventhal, 1935*).

Later a broad spectrum of clinical presentations came to be recognized and it is now clear that many women have polycystic ovaries in the absence of one or tow of the triad of hirsutism, obesity and menstrual irregularities. The name was changed from polycystic ovary syndrome to polycystic ovarian disease "PCOD" (*Goldzieher and Dizerega, 1985*).

Treatment swings between surgery and medical induction of ovulation. Those who support surgery argue that medical induction of ovulation has moderate results. The ideal agent for ovulation induction is not yet available. The ideal agent should be inexpensive, convenient to use, associated with a low incidence of side effects and no inherent anti-fertility effect. In addition, in

inducing normal ovulation, it should lead to a high conception rate, but low incidence of multiple pregnancy. (*Fox and Hull, 1989*).

A surgical approach to ovulation induction for PCOD holds many attractive advantages for patients over gonadotropin therapy because of cost in money, time and efforts (*Fox and Hull, 1989*). This line of therapy is extremely suitable to our patients.

Surgery as a line of treatment started by the intelligent observation of *Stein's group (1949)*, that diagnostic ovarian biopsy was often followed by commencement of ovulatory menstrual cycles in a substantial proportion of cases, and wedge resection of the ovaries was quickly adopted as a treatment of anovulatory infertility associated with PCOD.

This was supported by *Goldzieher and Green (1962)* who reviewed 1079 cases and reported that 80 percent developed regular menstrual cycles and 63 percent conceived after wedge resection of the ovaries in PCOD.

However this line of treatment received a set-back by *Buttram and Vaquero (1975)* who reported a high risk of pelvic adhesions formation and consequent tubal infertility.

To minimize the surgical trauma that results from ovarian wedge resection laparoscopic ovarian electrocautery was used as an alternative (*Gjonnaess, 1984*).

More recently it was shown that ovulation can be induced in patients with PCOD by partially vaporizing their ovaries with laser energy. Favourable results have been claimed without occurrence of pelvic adhesions. (*Daniell and Miller, 1989*).

There is no comparative study between the last two surgical methods of treatment, and because electrocautery equipment is available in any hospital while laser is a sophisticated expensive apparatus, comparative study between the two techniques is a logical and valuable piece of research.

If the results are the same, electrocautery will be much easier and cheaper and can be applied to any patient with PCOD in any hospital

THE AIM OF THE WORK

AIM OF THE WORK

The aim of this work is to evaluate and compare the results of treatment of patients with PCOD with laser vaporization and electrocautery of the ovaries as regards, the results of therapy, difficulties, morbidity and post-operative adhesions.

***REVIEW
OF
LITERATURE***

HISTORICAL REVIEW

The finding of a "Sclerocystic" change of the ovary was first recorded by *Chereau in 1844* and later in the century oophorectomy became a widely used therapeutic procedure. In 1896, *Waldo* applied the principle of partial resection rather than extirpation. In American literature, *Findley* described wedge resection for "cystic degeneration of the ovary" as early as 1904. *Forgue and Massabuau (1910)* reviewed the subject of sclerocystic ovaries and perhaps suggested the more appropriate name "microcystic ovaries".

In 1929 *Stein* performed his first wedge resection of bilateral polycystic ovaries for amenorrhea, since then he and his associates (*Cohen, Elson and Leventhal*) have reported on an accumulated group of patients who presented what *Stein* has termed a definite clinical syndrome. The existence of such a syndrome as a specific clinical entity has been questioned in recent years by several investigators, (*Goldzieher and Green 1962, and Taymor and Barnard, 1962*). These investigators are of the opinion that what has been designated as the *Stein-Leventhal* syndrome possibly represents either a number of specific entities or is simply one stage of a progressive disease. However, most investigators recognize the defined syndrome as a single entity within itself (*Barry and Irish, 1961*).

The triad of signs (amenorrhea, obesity and hirsutism) in the presence of bilateral polycystic ovaries is a sine qua non of the diagnosis of what was known as the stein-leventhal syndrome (*Stein, 1964*).

Subsequent morphological, biochemical and endocrinological investigations of the syndrome by numerous investigators revealed a heterogenous assay of underlying defects and this resulted in a serious debate even in the existence of such a well defined syndrome. Since the presence of bilateral enlarged multicystic ovaries was a pre-requisite for the diagnosis, the term polycystic ovarian syndrome was introduced a few years later to emphasize its heterogenicity (*Yen, 1980*).

Later, a broad spectrum of clinical presentations came to be recognized, and it is now clear that many women have polycystic ovaries in the absence of one or two of the triad of hirsutism, obesity and anovulation (*goldzieher, 1981*). It seems that the syndrome described by *Stein and Leventhal* characterises only a small fraction of a much larger population of patients with polycystic ovaries (*Goldzieher and Dizerga, 1985*), and so the name was changed from polycystic ovary syndrome to polycystic ovarian disease.

ETIOLOGY OF PCOD

The etiology of this disorder is still uncertain, how the vicious cycle is generated is difficult to explain.

Futterweit (1984 d) proposed that PCOD develops in two stages : a generating phase in which a factor or factors within or without the hypothalamic - pituitary - ovarian axis promote the initial disruption, and an effector stage in which the disturbance is propagated throughout the system and thereby amplified. Unfortunately the disease is propagated in such a way that neither the nature nor the site of action of the generating factor(s) is easily determined.

Franks et al. (1985) have concluded that PCOD is not one distinct nosological entity but rather that the polycystic ovary simply represents a non-specific response of the H.P.O. axis common to a number of stimuli. It has been reported that PCOD occur as an inherited disorder.

Most previous work has focused on detection and interpretation of endocrine abnormalities. The possibility of underlying aberrations of ovarian, hypothalamic, pituitary and adrenal function have been recently explored :