

**CLINICAL PRESENTATION, ULTRASONIC,  
LAPAROSCOPIC EXAMINATION AND LH : FSH  
RATIO IN POLYCYSTIC OVARIAN DISEASE**

**THESIS**

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DEGREE IN OBSTETRICS AND GYNAECOLOGY

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

وَقُلِ الْحَمْدُ لِلَّهِ سِيرِكُمْ ءَايَاتِهِ فَتَعْرِفُونَهَا  
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# INTRODUCTION

**INTRODUCTION**  
**HISTORICAL BACKGROUND**

Polycystic ovarian disease PCOD is a common gynaecological endocrine disorder (Rojanasakul et al., 1989).

It is a condition with exaggerated steady state of tonic gonadotropin and oestrogen function associated with persistent anovulation (Speroff et al., 1983). This steady state is only relative as pregnancy can occur without treatment.

Gross sclerocystic changes in the human ovary were clearly described by Chereau in 1845, but more interest was aroused in 1935, when this anatomical abnormality was related by Stein and Leventhal to a clinical syndrome consisting of "menstrual irregularity featuring amenorrhea, a history of sterility, masculine type hirsutism, and obesity" (Stein and Leventhal, 1935).

The diagnosis of this disorder relies mostly on the clinical presentation and hormonal profiles of the patient (Yen, 1980 and Goldzieher, 1981) Laparoscopy and laparotomy had been used to confirm the diagnosis (Rojanasakul et al., 1989). The development of high resolution real-time ultrasound during the past decades leads to the new approach for ovarian visualization (Hackeloer et al., 1979; Hackeloer and Nitschke-Dabelstein, 1980).

In contrast to laparoscopy that only the external surface of the ovary could be seen, ultrasonic examination allow a direct visualization of the internal structure of the ovaries. It has now replaced laparoscopy as the method for assessing the ovarian morphology in many clinical conditions (Rojanasakul et al., 1989).

# AIM OF THE WORK

#### **AIM OF THE WORK**

The aim of this work is to study the ultrasonic appearance of suspected polycystic ovaries and to correlate it to the different clinical, hormonal and laparoscopic findings of the polycystic ovarian disease.

# REVIEW OF LITERATURE

#### ANATOMIC PATHOLOGY OF POLYCYSTIC OVARIAN DISEASE (PCOD)

In the classic description of PCOD the ovaries were bilaterally enlarged with a smooth, pearly looking surface. The capsule appeared thickened and sclerotic, below which were multiple subcapsular cysts (Goldzieher, 1981).

It is now understood that in some instance the appearance of the ovaries may not conform to the original description and may be atypical as normal sized ovaries have been observed in patients with PCOD (Cheung and Chang, 1990).

However, in seemingly normal women with regular ovulation and menstrual cyclicity, multiple cystic follicles in both ovaries have been identified (Polson et al., 1988). Thus to make the diagnosis of PCOD based on ovarian morphology alone may not be necessarily accurate. Further investigation of the relationship between ovarian anatomy and ovulatory function is clearly warranted (Fox and Hull, 1989). The ovarian capsule is usually thickened and has been reported to be 0.2-0.4 mm in 65% of patients undergoing wedge resection (Cheung and Chang, 1990).

Immediately below the epithelial layer there are thickened collagen fibres that extend into and merge with the cellular outer cortical stroma. No correlation has

been found between the thickness of the capsule and the morphology of ovarian structures (Cheung and Chang, 1990). Capsular thickness also varies in the same ovary. It has been suggested that the thickening process is in response to chronic ovarian distension (Hugesdon, 1982). Alternatively, the sclerotic changes may merely reflect the result of hyperandrogenism as suggested by studies performed in animals (Goldzieher, 1981). The abnormal follicles are characterized by relative lack of granulosa cells leading to a reduced total complement of cells surface FSH receptor (Cheung and Chang, 1990).

In addition, mitotic activity in these cells is decreased. On examination under electromicroscopy, the granulosa cells exhibited abundant rough-surface endoplasmic reticulum in association with a large number of polyribosome. The Golgi apparatus is well developed as are mitochondria with lamellar cristae. These features are consistent with active protein rather than steroid production (Cheung and Chang, 1990).

In contrast, the theca cell layer is more dense and proportionately thicker than that of the granulosa cells. The ultrastructure of the thecal cell is characterized by abundance of smooth-surface endoplasmic reticulum, lipid droplet and mitochondria with tubular cristae, feature typical of steroid production (Cheung and Chang, 1990).

Collectively, these observations indicate that polycystic ovaries are definitely abnormal with respect to gross morphology, histology, and electromicroscopy.

The ultrastructural characteristics of thecal cells are consistent with the increased production of ovarian androgen. That granulosa cells display findings consistent with active protein synthesis raises the issue of a role for small ovarian peptides in the pathophysiology of this disorder (Cheung and Chang, 1990).