

GLUCOSE / INSULIN RATIO AND LUTEINIZING HORMONE PRODUCTION IN POLYCYSTIC OVARIAN DISEASE

Thesis

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in Gynaecology and Obstetric



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INTRODUCTION

AND

AIM OF THE WORK

Historical review and definition :

Gross sclerocystic changes in the human ovary were described by Cheseau, 1845, and partial resection of such ovaries was being practiced before 1897 in Europe by Gusserow, Martin, Wiedow, Zweifel, and others. More interest was aroused in 1935 when this anatomical abnormality was related by Stein and Leventhal to a clinical syndrome consisting of menstrual irregularity, amenorrhea, history of sterility, hirsutism, and retarded breast development and obesity (Goldzieher et al., 1981).

This syndrome is called polycystic ovarian disease (PCOD) which is a complex state of chronic anovulation due to chronic hormonal imbalance involving inappropriate gonadotropin production, with relative excessive secretion of luteinizing hormone (LH) to follicular stimulating hormone (FSH) resulting in an increase in ovarian androgen production (Dunaif et al., 1989).

The end result of this state of hormonal imbalance will be obesity, hirsutism, bilateral polycystic ovaries and anovulation (Bernstein et al., 1991). Hyperinsulinaemia and insulin resistance are important features of this disease (Shelly and Dunaif, 1990).

The obese women with polycystic ovarian disease (PCOD) were showed a decreased insulin sensitivity (Rajkhowa et al., 1994).

The aetiology of the polycystic ovarian disease (PCOD) is unknown, but may be transmitted as autosomal dominant, X-linked trait, or endocrinal disturbance in the hypothalamo- pituitary- ovarian- adrenal axis or enzyme defects (Speroff, et al., 1988).

Aim of the work

This study was designed to assess the value of measurement of glucose- insulin ratio and LH production in relation to pathogenesis of patients with polycystic ovarian disease (PCOD).

REVIEW

OF

LITERATURE

