ARTICLE / INVESTIGACIÓN

Evaluating the clinical significance of RBP4, PAI-1, and some trace elements in women with Polycystic Ovary Syndrome

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Abstract: To assess and compare clinical, hormonal, and metabolic factors with blood levels of RBP4, PAI-1, and trace elements in women with and without polycystic ovarian syndrome (PCOS). A cross-sectional clinical investigation was undertaken. From December 2020 until January 2022, samples were taken at the Basrah Hospital for Obstetrics and Children's infertility center. Significant changes ($p< 0.05$) were in HOMA-IR, E2 and Ts. Levels of PAI-1, RBP4, AMH, LH, LH/FSH, PRL and Cu were significantly ($p<0.01$) increased, and levels of Se, Zn, Mg and E2/T were significantly ($p<0.01$) decreased, between the patient (1o PCOS and 2o PCOS) and control groups, the QUICKI level did not differ significantly ($p>0.05$). Compared to the control group, FSH levels were especially ($p<0.05$) higher in non-obese PCOS patients and lower in obese PCOS patients. Area under the receiver operating characteristics (ROC) curve (AUC) results indicate RBP4 and PAI-1 may be more effective predictors biomarkers for PCOS in expectant women. While trace elements might be considered a protective factor in the emergence of PCOS, metabolic abnormalities and IR in PCOS-affected individuals are associated with the levels of RBP4 and PAI-1, which appear to be a more acceptable diagnostic marker in the early prediction of PCOS.

Key words: Polycystic Ovary Syndrome, RBP4, PAI-1, Trace elements.

Introduction

A diverse hormonal and metabolic condition with only a partially understood pathophysiology, polycystic ovarian syndrome (PCOS) is the most prevalent endocrinopathy among women of reproductive age, with a frequency of up to 15%. In contrast, patients with PCOS who lost 5-7 % of their basal weight showed improvements in insulin resistance, hyperinsulinemia, and hyperandrogenism, which appear to be key factors in at least some causes of PCOS pathogenesis. Therefore, the pathogenesis of PCOS in women may still be largely unknown. Still, it has primarily been linked to increased anti-Mullerian hormone (AMH) levels brought on by oligo-ovulatory cycles or a folliculogenesis disorder, which results in increased preantral and small antral follicle counts[1].

Insulin resistance is a biological misunderstanding in which the body's insulin hormone receptors on cell membranes do not respond to insulin as intended, preventing blood glucose from entering cells and causing a hypoglycemic reaction. Reduced insulin hormone's capacity to control and signal changes in glucose levels in the blood may lead to insulin resistance due to the pancreas pumping out high insulin dosages to get the glucose out of circulation and into cells[2].

Whether PCOS or concomitant obesity is to blame for insulin resistance is still debatable. Any adipose tissue dysfunction may be the primary source of the observed IR and, as a result, the metabolic and cardiovascular effects of the illness. Additionally, some studies suggest that PCOS may cause changes in adipocyte function that affect adipokine release[1].

Retinol-binding protein 4 (RBP4) is an adipokine released by the liver and adipose tissue and is a member of the lipocalin protein family. Its primary function is transporting vitamin A (retinol) from the liver to the peripheral tissues. By attaching to cell surface receptors or acting through retinoic acid on retinoic acid receptors and retinoic acid-X receptors, RBP4 can affect peripheral tissues[3]. Human plasminogen activator inhibitor-1 (PAI-1) is an inhibitor of tissue-type and urokinase-type plasminogen activators (tPA and uPA), which turn plasminogen into plasmin. Because of its capacity to suppress the fibrinolytic activity of tissue-type plasminogen activator (tPA), which produces active plasmin from plasminogen and subsequently eliminates fibrin, PAI-1 is a key regulator of the endogen. Also, it is an essential member of the serine protease inhibitor superfamily, known as Serpin E-1; synthesized by many tissue and cell types, free PAI-1 is relatively inactive in its free form and readily converts into its latent state[4].

The present study examined and compared the association of insulin resistance with mentioned adipokines and trace elements as a clinical predictor for the development of POCS among obese and non-obese women in Basrah province (southern Iraq).

Materials and methods

Study Design and Subjects Recruitment

This study is a clinical case-control trial. Samples were

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