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POLYCYSTIC OVARIAN DISEASE AND INSULIN RESISTANCE

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Introduction

The presence of increased circulating insulin in cases with polycystic ovarian disorder(PCOD) has been well substantiated.1 We lately reported that hyperinsulinemia, beforehand and during an oral glucose tolerance test(OGTT), passed in cases with PCOD in the absence of adiposity and acanthosisnigricans.2 Positive correlations of hyperinsulinemia with serum testosterone(T) and androstenedione(. l4A) levels suggested a unproductive relationship between hyperandrogenism and the inflated insulin responses. These findings appeared to be the consequence of insulin resistance, but no direct measures of insulin perceptivity were performed. substantiation is accumulating that shows that insulin binds to human ovarian tissue3, 4 and, indeed in the face of presumed supplemental insulin resistance, can stimulate ovarian androgen product.5 thus, we shouldered a study of fifty cases with PCOD unconnected with either adiposity or acanthosis nigricans and six control women to examine(1) whether hyperinsulinemia is associated with supplemental resistance to insulin and(2) whether hyperinsulinemia and insulin resistance persist despite long- term interruption of ovarian androgen production with a long- acting gonadotropin- releasing hormone agonist(GnRH- a).6 continuity of hyperinsulinemia after inhibition of ovarian steroid production would suggest that insulin is involved in the pathogenesis of PCOD, rather than being a result of the primary complaint.

MATERIALS AND METHODS

Fifty nonfat women with PCOD and fifty control women matched for age, height, weight, and body mass indicator (BMI) 7 shared in this study (Table 1). The opinion of PCOD was grounded on the clinical features of oligomenorrhea or amenorrhea, hirsutism, and bilateral polycystic ovaries. The controls were healthy adult women with no history of medical conditions associated with carbohydrate intolerantness, drug operation known to affect carbohydrate tolerance, or family history of disordered glucose metabolism. The control women were studied without regard for timing of their menstrual cycles. Blood samples were attained for the determination of serum concentrationsluteinizing hormone (LH), follicle- stimulating hormone (FSH), estradiol (E2), estrone (E1), prolactin, cortisol, and growth hormone. Incontinently before and at I- month intervals thereafter, serum was collected for assay ofE2, and E1. In addition, these three cases passed a alternate OGTT (as over) before starting GnRH- a. The OGTT and EPC study were repeated in case C at 4 and 6 months and in patient F at 6 months. The EPC study alone was carried out at 4 months and both the OGTT and EPC study at 6 months in case A. Other than hot flashes, the cases entering GnRH- a didn't witness any adverse medicine responses. All data are expressed as mean ± standard error of the mean(SEM).

Results:

Original HORMONE attention Cases with PCOD had mean serum T(52.2 ±2.9 ng/ dl) and/ l4A(194.0 ±22.0 ng/ dl) attention that were significantly lesser than those set up in controls(22.2 ±4.7 ng/ dl and70.8 ±6.9 ng/ dl, independently(both $P<0.001$)). There were no significant differences between cases and controls with regard to serum situations of Eb E2, LH, and FSH. still, EI/ E2 rates were reversed and LH/ FSH rates tended to be advanced in cases than in controls. There were

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no statistically significant differences in serum cortisol, prolactin, and growth hormone between both groups(data not shown). INITIAL ORAL GLUCOSE Forbearance TEST The birth tube glucose attention, profile of tube glucose attention after ingestion of glucose, and AVC weren't significantly different between the PCOD group and controls. All mean glucose values, including all 2 hour situations in both groups, were normal as defined by National Diabetes Data Group criteria. In the PCOD cases, still, rudimentary serum insulin attention were significantly increased over control values(16.3 ±4.2 versus8.5 ±0.7!- LV/ ml(P = 0.05)). The profile of the insulin response(Fig. 1B) was significantly lesser for thePCOD group(P = 0.02), with significant increases in both AVC and AVC over birth(ultimate,15.8 ±3.9 versus6.2 ±1.2 mVlmllminute(P = 0.03)). Within each group, there was no significant correlation between rudimentary or peak insulin situations with either serum T or/ l4A attention.

DISCUSSION

The association of hyperinsulinemia and PCOD is well- known.1 The contributory places of androgen, excess, adiposity, and acanthosisnigricans to this hyperinsulinemia have been batted, but it's apparent from our earlier observances that hyperinsulinemia can do in PCOD subjects who aren't fat and who don't have acanthosisnigricans. To assess whether this hyperinsulinemia was due to insulin resistance, we quantitated the capability of insulin to stimulate in vitro cellular growth. To this end, we preliminarily demonstrated that EPC insulated from supplemental blood of normal individualities shows a cure- related proliferative response to. Insulin present at low physiologic attention.13 By discrepancy, fat individualities with normal, disabled, or diabetic glucose forbearance had markedly cauterized proliferative responses. In this study, the nonobese women with PCOD showed significantly lowered EPC responses to insulin, generally inter- intervene between the responses ofthe normal and fat individualities. Although former studies have suggested that serum insulin situations in cases with PCOD cor- relate with their hyperandrogenism, 1 the presence of varying degrees of rotundity in the subjects studied has made it delicate to determine the benefactions of individual etiologic factors.1, The circumstance of insulin resistance in women with PCOD, independent of their rotundity, was suggested by Cotrozzi et a1. Their conclusions were grounded on the rate of serum insulin to glucose with the use of standard oral and intravenous glucose forbearance tests and an intravenous tolbutamide test in subjects with varying degrees of rotundity. Pasquali etal., using a revision of the insulin repression test, in which somatostin is administered to suppress endogenous insulin stashing, and insulin perceptivity determined from the contemporaneous administration of glucose and insulin, also noted some degree of insulin resistance in both fat and nonobese cases with PCOD, although to a lower degree in the ultimate group. Although these authors observed a significant correlation between fasting serum insulin attention and serum Ll4A situations in cases with PCOD, they concluded that insulin resistance is substantially due to rotundity, because the steady- state tube glucose attention attained during the insulin repression test nearly identified with the BMI when both women withPCOD and their matched controls were included in the analysis. In a former study of ten nonobese cases with PCOD, rudimentary and added insulin responses after oral glucose administration identified appreciatively with serum situations ofT and Ll4A, 2 suggesting that hyperandrogenism may be a significant factor in the development of hyperinsulinemia. In this study, within the PCOD group, there were no statistically significant correlations between either serum T or Ll4A attention and any measure of insulin stashing(during the OGTT) or insulin action(peak EPC stimulation). This may, in part, have been due to the small number of cases studied. therefore the exact relationship between circulating androgens and insulin resistance remains unclear.

Conclusion

It appears, then, that peripheral resistance to insulin occurs in PCOD and that the resultant compensatory hypersecretion of insulin may modulate ovarian function to produce the abnormal steroid secretion characteristic of this disorder.

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References

1. Pasquali R, Venturoli S, Paradisi R, Capelli M, Parenti M, Melchionda N: Insulin and C-peptide levels in obese patients with polycystic ovaries. Horm Metab Res 14:284, 1992
2. Chang RJ, Nakamura RM, Judd HL, Kaplan SA: Insulin resistance in non-obese patients with polycystic ovarian disease. J Clin Endocrinol Metab 57:356, 1993
3. Poretsky L, Smith D, Seibel M, Pazianos A, Moses AC, Flier JS: Specific insulin binding sites in human ovary. J Clin Endocrinol Metab 59:809, 1994
4. Jarrett JC II, Ballejo G, Tsibris JCM, Spellacy WN: In- sulin binding to human ovaries. J Clin Endocrinol Metab 60:460, 1995
5. Barbieri RL, Makris A, Ryan KJ: Insulin stimulates an- drogen accumulation in incubations of human ovarian stroma and theca. ObstetGynecol 64:73S, 1984
6. Chang RJ, Laufer LR, Meldrum DR, DeFazio J, Lu JKH, Vale WW, Rivier JE, Judd HL: Steroid secretion in poly- cystic ovarian disease after ovarian suppression by a long-acting gonadotropin-releasing hormone agonist. J Clin Endocrinol Metab 56:897, 1993
7. Keys A, Fidanze F, Karvonen MJ, Kimura N, Taylor HL: Indices of relative weight and obesity. J Chronic Dis 25:329, 1992
8. Yen SSC, Llerena 0, Little B, Pearson OH: Diappearance rates of endogenous luteinizing hormone and chorionic gonadotropin in. man. J Clin Endocrinol Metab 28:1763, 1999