AEPCOS QUARTERLY PUBLICATION LIST

OCTOBER—DECEMBER 2014

Highlighted articles

Calderon-Margalit R, Siscovick D, Merkin SS, Wang E, Daviglus ML, Schreiner PJ, Sternfeld B, Williams OD, Lewis CE, Azziz R, Schwartz SM, Wellons MF. Prospective association of polycystic ovary syndrome with coronary artery calcification and carotid-intima-media thickness: the Coronary Artery Risk Development in Young Adults Women's study. Arterioscler Thromb Vasc Biol. 2014 Dec; 34(12):2688-94 ***(KMH)***

There remains debate about the cardiovascular impact of a diagnosis of PCOS. While there is evidence that different phenotypes of PCOS exhibit different metabolic risk, there are few well characterized longitudinal studies to assess cardiovascular risk in PCOS. The Coronary Artery Risk Development in Young Adults (CARDIA) study is a population based that was initiated in 1985-6 and included 5115 adults aged 18-30 years in the US. The population was 54% women. Serum was stored at year 2 of the study to measure androgens. At year 16 of the study 1163 women completed reproductive health questions. Women were defined as oligomenorrheic based on cycle irregularities age 20-30 and hyperandrogenism was defined by elevated testosterone at year 2 of the study in women not on oral contraceptives or pregnant, or by development of hirsutism. In year 20 of the study coronary artery calcium (CAC) and carotid intima media thickness (IMT) studies were performed on 982 women. The mean age was 45.3. This included 55 women with PCOS by oligomenorrhea and hyperandrogenisn, 156 women with hyperandrogenism alone, 103 with oligomenorrhea alone and 668 with neither. CAC was present in 10.3% of controls, 11.7% of isolated oligomenorrheaic patients, 6.4% with isolated hyperandrogenism and 23.6% of PCOS, resulting in a multivariable adjusted OR of 2.70 (1.31-5.60) for CAC in PCOS. HOMA-IR was increased in PCOS compared to the other groups. BMI was not different between the groups with a mean BMI of 29.3 in the PCOS women. Women with PCOS had increased bulb or internal carotid artery IMT. Therefore this study demonstrates that there appears to be an increased rate of subclinical cardiovascular disease in women at age 45 with a history of PCOS by characteristics present in their 20s. Neither oligomenorrhea alone nor hyperandrogenism alone was associated with a similar risk. Overall this study is limited by the generally small number of women diagnosed with PCOS (55) by these criteria and the menstrual data was by delayed self-report. There are no ultrasound data to assess the contributions of this component of the phenotype. The outcome measure if one of subclinical disease and therefore it cannot answer a question regarding cardiovascular morbidity. However this is a population based cohort with a multicentre design

and 20 years of follow up. As this was a nonreferral population with diagnostic criteria applied similarly across the board, the population is not biased by the concern of more significant disease presenting for diagnosis. This study provides additional support for continuing study to assess the cardiovascular disease risk of PCOS.

Kurzthaler D, Hadziomerovic-Pekic D, Wildt L, Seeber BE. Metformin induces a prompt decrease in LH-stimulated testosterone response in women with PCOS independent of its insulin-sensitizing effects. Reprod Biol Endocrinol. 2014 Oct 11;12:98 *(CM)*

This study provides additional clinical evidence about the action of metformin on androgen production independent of its metabolic effect. The mechanisms by which metformin exerts its effects in PCOS are not totally understood. There is scarce evidence of a direct effect of metformin on ovarian steroidogenesis independent of its effects on insulin sensitivity. In vitro studies using cultured ovarian cells have demonstrated a direct metformin-effect on ovarian steroidogenesis. A decrease in serum free T has been shown following metformin in PCOS, thought to be secondary to a reduction in insulin secretion. Most previously published studies reporting improvement in hyperandrogenemia have evaluated androgen levels in PCOS women following 8 to 12 weeks of metformin, though some have observed a reduction in T within one week of treatment. The primary aim of this study was to test the hypothesis of a direct effect following 2 days of metformin on the ovary, independent of improving insulin sensitivity in women with PCOS, evaluating short-term changes in androgen levels and LH-stimulated androgen response. The secondary aim was to determine the longer-term effects during a 12 week follow-up with metformin on clinical, metabolic and endocrine parameters, including changes in anthropometric measures, insulin sensitivity, menstrual pattern, metabolic and endocrine parameters.

Patients (n = 19) were 18 - 40 years old with PCOS, as defined by the Rotterdam criteria. After the baseline assessment, an LH stimulation test was performed with a load of SC 75 I.U. recombinant human LH. Blood samples were collected at −15, 0, 15, 30, 45 and 60 minutes before and after LH administration. Androgens and estradiol were measured before LH and repeated at each time of assessment during the LH stimulation test. Patients were divided into those who received metformin 500 mg, three times daily (n = 10) and those who received placebo in similar form (n = 9) for the following 2 days. The authors evaluated the short-term effects of metformin compared to placebo on basal and LH- stimulated androgen secretion and on hormonal and metabolic parameters during a randomized, double-blinded placebo-controlled clinical trial. The serial values obtained during the 3-h OGTT were used to calculate the area under the curve for insulin and glucose. In addition, they investigated the effects of metformin on ovulation, metabolic and endocrine parameters in a 3 month follow-up evaluation.

They found that compared to placebo, 2 days of metformin was associated with a borderline significant reduction in the free androgen index ($p = 0.05$), with a reduction in the serum LH-stimulated T ($p = 0.03$). Following 3 months of use, a decline in T ($p = 0.04$) and an increase in DHEAS ($p = 0.03$) was observed, independent of changes in weight, metabolic parameters or insulin sensitivity. The findings therefore suggest that there is a direct effect of metformin on androgen production at the ovarian level, independent of its insulin sensitizing effects. However, the effect on menstrual pattern and the rate of ovulation after the 3 months of metformin use was not completely reported.

The conclusion of the study is that metformin induces a prompt decrease in LH-stimulated T secretion after only 2 days of use in women with PCOS. This action precedes the effects of metformin on insulin sensitivity or weight loss.

LIST OF PUBLICATIONS

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Ahmad IC, Yilmaz TF, Kocakoç E. Doppler ultrasonography and magnetic resonance imaging findings of testicular adrenal rest tissue in a patient with 11 β hydroxilase deficiency. Case report. Med Ultrason. 2014 Dec;16(4):383-5. PMID: 25463895.

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PCOS-Adolescence

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PCOS-Dermatology

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PCOS-Endocrine Disruptors

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