[5]College of Kinesiology, Physical Activity Complex, University of Saskatchewan, Saskatoon, SK, Canada.

Women with polycystic ovary syndrome (PCOS) exhibit reduced skeletal muscle insulin-mediated glucose uptake. Altered muscle mass may affect insulin resistance (IR) and inflammation, thereby potentially aggravating reproductive status including ovulatory cyclicity and fertility potential. However, the relationship between PCOS and skeletal muscle mass is elusive given conflicting reports on protective or detrimental influence of PCOS endocrine derangements (hyperandrogenism, IR) on muscle. We evaluated whether muscle mass and function are affected by PCOS in response to a call to elucidate musculoskeletal alterations in the International Evidence-based Guideline for the Assessment and Management of PCOS. Databases of MEDLINE, Web of Science, and Scopus were searched (January 1990 to September 2020) to identify observational studies on skeletal muscle mass (lean tissue mass) and function (strength) in PCOS and control groups. The primary outcome was total lean body mass (LBM) or fat-free mass (FFM). Data were pooled by random-effects models and expressed as weighted mean differences and 95% confidence intervals. Forty-five studies (n = 3,676 [1,854, PCOS; 1,822, controls]) were eligible. Forty-one evaluated lean tissue mass and five strength. PCOS groups had increased total (0.83 [0.08, 1.58] kg; P=0.03; I^2 = 72.0%) yet comparable trunk (0.84 [-0.37, 2.05] kg; P = 0.15; I^2 = 73.0%) LBM/FFM. There were no associations between mean differences of groups in total testosterone (TT) or homeostatic model assessment of IR (HOMA-IR) and total/trunk LBM/FFM (All: P ≥ 0.75) by meta-regressions. However, mean differences of groups in body mass index (BMI) were associated with total (0.65 [0.23, 1.06] kg; P < 0.01; I^2 = 56.9%) and trunk (0.56 [0.11, 1.01] kg; P = 0.02; I^2 = 42.8%) LBM/FFM. Accordingly, PCOS sub-group with overweight/obesity (BMI ≥ 25 kg/m^2) exhibited greater total LBM/FFM than controls (1.58 [0.82, 2.34] kg; P < 0.01; I^2 = 64.0%) unlike a lean (BMI < 25 kg/m^2) sub-group (-0.45 [-1.94, 1.05] kg; P = 0.53; I^2 = 69.5%). Some study results were contradictory (i.e., increased appendicular mass or strength in PCOS group or comparable findings between groups) and study methodology varied; thus, inclusion in meta-analyses was not possible. PCOS cohorts have a tendency for increased total and trunk lean tissue mass likely attributed to obesity. However, most critically, whether PCOS influences other lean tissue areas (appendicular), morphology, and function is unclear. Our observations do not support any protective/detrimental influence of hyperandrogenism (TT) or IR (HOMA-IR) on lean mass. Heterogeneity among studies warrants research to address any contributions of lifestyle, healthcare, and biological factors to observed differences for future guideline recommendations to improve PCOS musculoskeletal and reproductive health (www.crd.york.ac.uk/PROSPERO ID, CRD42020203490).

Reproductive Endocrinology

HYPERANDROGENIC DISORDERS THROUGHOUT THE LIFESPAN AND INTO THE NEXT GENERATION

Sleep Characteristics Among Women With and Without PCOS

Karen Oppermann, PhD,
Stéfanie Zamboni Perozzo Hemkemeier, Graduate school,
Ana Victoria Reichert, Graduate school, Lais Weber,
Graduate school, Laura Rinaldi, Graduate school.
University of Passo Fundo, Passo Fundo, Brazil.

Clinical studies indicate that sleep disorders, including obstructive sleep apnea (OSA) and excessive daytime sleepiness, occur more frequently among women with PCOS compared to comparison groups without the syndrome. The presence of OSA in PCOS is associated with worsening of metabolic parameters. There is some evidence that obesity directly contributes to OSA among women with PCOS, although, it does not fully account for findings from community-and clinic-based studies. Questionnaires are used as screening for sleep disorders. The objective was to verify the quality of sleep, the prevalence of OSA and daytime sleepness among women with PCOS compared to control group. The sample size calculation was based on estimates bad quality of sleep among women with PCOS in 80% and among control women in 45% (1). The sample with 58 women, 29 each group, had a power of 80%, with a significance level of 0.05. This is a cross sectional study with 29 patients with PCOS and 31 controls from Gynecology Endocrinology Ambulatory of São Vicente de Paulo Hospital, Passo Fundo, RS, Brazil, who consulted between January 2017 and March 2020. Women with PCOS by Rotterdam criteria and controls were under 40 years old and no pregnant. Controls women had regular cycles, no history of PCOS or hirsutism and had normal ovaries on transvaginal ultrasound. Age, BMI, blood pressure (BP), waist circumference (WC) were measured. It was applied the validated questionnaires of Pittsburgh Sleep Quality Index, to classify in good and bad sleep quality; Epworth Sleepiness Scale for daytime sleepiness and Berlim Questionnaire for evaluate sleep apnea risk. The mean of age was 30.6 ± 5.9, PCOS 29.1± 6.7 versus 32.3±4.7, p=0.06. The group of PCOS women was heavier (BMI 32.4±6.1versus 28.0 ± 5.3, p=0.04) and presented higher WC (101.3±16.1 versus 91.6 ±14cm, p=0.03). The mean of BP was similar between the groups. The prevalence of bad sleep quality was 53.6% for women with PCOS and 63.1% for controls (p=0.29). The daytime sleepiness was present in 14,5% of the women with PCOS and 35.7% of controls (p=0,061) and the sleep apnea risk was 32.1% for women with PCOS and 21.4% for controls (p=0.27). The association of risk of OSA was verified with robust multivariate Poisson. The prevalence ratio (PR) of BMI ≥ 30 was 1.820 (CI 1.281-2.587) p<0.001, BMI ≥ 25 1.549 (IC 1.067- 2.250) p=0.02, adjusted for age, WC and PCOS diagnosis. In conclusion, there was no difference in prevalence of quality of sleep, OSA risk or daytime sleepiness between women with PCOS and controls. The risk of OSA was higher in obese women independently of age, abdominal circumference and PCOS diagnosis.

Reference: (1) Fernandez et al., Nature and Science of Sleep 2018; 10: 45–64.

Reproductive Endocrinology

HYPERANDROGENIC DISORDERS THROUGHOUT THE LIFESPAN AND INTO THE NEXT GENERATION

The Association Between Clinical and Biochemical Hyperandrogenism in Women With Female Pattern Hair Loss

Samih A. Odhaib, MD[1], Abbas A. Mansour, MD, FRCP, FACE[2], Khalil I. Al Hamdi, Ph.D., FRCP[3].
[1]Faihaa Specialized Diabetes Endocrine and Metabolism Center, College of Medicine, University of Basrah, Basrah, Iraq, [2]Faiha Specialized Diabetes Endocrine and Metabolism Center, College of Medicine, University of Basrah, Basrah, Iraq, [3]Department of Medicine, College of Medicine, University of Medicine, Basrah, Iraq.

Background: The exact association between clinical and biochemical hyperandrogenism (HA) is heterogeneous and cannot be ascertained, especially in normoandrogenic women. **Aim:** Evaluate any association between clinical HA phenotypes and biochemical parameters in premenopausal women with female pattern hair loss (FPHL). **Methods:** A cross-sectional observational study on 362 women with different degrees of FPHL, who were assessed for general characteristics, the degree of FPHL by Sinclair's score, hirsutism by modified Ferriman-Gallwey (mFG) score. Evaluation for biochemical HA included total testosterone (TT), sex-hormone-binding globulin (SHBG), calculated free testosterone (FT), calculated bioavailable testosterone (BT), and dehydroepiandrosterone sulfate (DHEA-S). The variables of clinical HA which were used in this study are FPHL, hirsutism, and acne. We used the Free and Bioavailable Testosterone Calculator to calculate the FT and BT. **Results:** The enrolled young premenopausal women's age range was (14-47 years). Around 78% of them were overweight or obese. Eighty-percent of women had a mild FPHL, with a median duration of three years where 2/3 of women had a duration < 3 years, and had no significant relationship to FPHL degree. About 73% of women had either a mild to moderate hirsutism, and around 16% had acne. The biochemical HA was confirmed in around 52% of women (n=188), who show high levels of calculated FT. The calculated BT is high in 78.5% of the enrolled women (n=284). The means of biochemical indicators for HA were in their reference ranges or slightly above, with no specific change pattern with the corresponding FPHL severity. None of these parameters had a significant relationship to the severity of FPHL. The duration of FPHL was not affected by any presumed variable of clinical or biochemical HA. **Conclusions:** FPHL severity is associated with other clinical HA signs like hirsutism and acne, but not to HA's biochemical parameter. Other parameters, like SHBG, HOMA-IR, and BMI, had no significant relation to the severity of FPHL. **Clinical implications:** FPHL severity does not correlate with the magnitude of hyperandrogenism. The assessment of women with FPHL is primarily clinical. The biochemical picture assists the diagnostic process.

Reproductive Endocrinology

HYPERANDROGENIC DISORDERS THROUGHOUT THE LIFESPAN AND INTO THE NEXT GENERATION

The Major Impact of Obesity on the Development of Type 2 Diabetes (T2D) in Women With PCOS: A Systematic Review and Meta-Analysis of Observational Studies.

Panagiotis Anagnostis, MD, PhD[1], Rodis Paparodis, MD[2], Julia Bosdou, MD, PhD[3], Christina Bothou, MD Msc[4], Dimitrios G. Goulis, MD, PhD[1], Djuro P. Macut, MD, PhD[5], Andrea Elizabeth Dunaif, MD[6], Sarantis Livadas, MD, PhD[7].
[1]Unit of Reproductive Endocrinology, [1]st Department of Obstetrics and Gynecology, Medical School, Aristotle University of Thessaloniki, Thessaloniki, Greece, [2]Center for Diabetes and Endocrine Research, University of Toledo College of Medicine and Life Sciences, Toledo, OH, USA, [3]Unit for Human Reproduction, [1]st Department of Obstetrics and Gynecology, Medical School, Aristotle University of Thessaloniki, Thessaloniki, Greece, [4]Klinik für Endokrinologie, Diabetologie und Klinische Ernährung, Universitätsspital Zürich, Zürich, Switzerland, [5]Clinic for Endocrinology, Diabetes and Metabolic Diseases, Faculty of Medicine, University of Belgrade, Belgrade, Serbia, [6]Icahn School of Medicine at Mount Sinai, New York, NY, USA, [7]Endocrine Unit, Athens Medical Center, Athens, Greece.

Background/Aims: Polycystic ovary syndrome (PCOS) is associated with disordered carbohydrate metabolism and an increased risk for T2D. However, there are limited data on the magnitude of this risk. Furthermore, 50-80% of women with PCOS are obese and obesity is known to have a synergistic deleterious effect on glucose tolerance in affected women. We systematically reviewed the literature regarding the association between PCOS, obesity and T2D risk. **Methods:** A comprehensive search was conducted in PubMed, CENTRAL and Scopus databases. Data are expressed as relative risk (RR) with 95% confidence intervals (CI). The I^2 index was employed for heterogeneity. The available data, did not allow us to analyze the impact of weight status as normal, overweight and obese and as a consequence the studied subjects were stratified as obese (BMI>30 kg/m^2) and non-obese (BMI<30kg/m^2). **Results:** Twelve studies fulfilled eligibility criteria, yielding a total of 224,284 participants (45,361 PCOS and 5,717 T2DM cases). Women with PCOS had a higher risk of T2D compared with to unaffected women (RR 3.13, 95% CI, 2.83-3.47, p<0.001; I^2 40.1%). When women with PCOS were stratified according to the presence or absence of obesity, the RR for developing T2D in obese compared with non-obese women with PCOS was 4.20 (95% CI 1.97-9.10; p<0.001). Moreover, compared to control women, the RR for developing T2D was significantly increased only in obese PCOS, RR 4.06 (95% CI 2.75-5.98; p<0.001). There was a trend toward significantly increased risk in non-obese PCOS women [RR 2.68 (95% CI 0.97-7.49; p=0.06). **Conclusion:** Women with PCOS have a >3-fold increased risk of T2D compared to women without PCOS, but this risk is substantially increased by the presence of obesity. Accordingly, weight reduction should be pursued in these women. **References:** 1. Dunaif A, Segal KR, Futterweit W, Dobrjansky A. Profound peripheral insulin resistance, independent of obesity, in polycystic ovary syndrome. Diabetes. 1989;38(9):1165-1174.2. Legro RS, Kunselman AR, Dodson WC, Dunaif A. Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective, controlled study in 254 affected women. J Clin Endocrinol Metab. 1999;84(1):165-169.3. Ehrmann DA, Barnes RB, Rosenfield RL, Cavaghan MK, Imperial J. Prevalence of impaired glucose tolerance