Home Based Aerobic Training and the Changes in Adipsin Levels and Visceral Adiposity Index (VAI) in Women with Polycystic Ovary Syndrome

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Abstract

Background: Polycystic ovary syndrome (PCOS) is prevalent in 5% - 8% of women of reproductive age and is one of the most important causes of infertility.

Objectives: The aim of this study was to investigate the effect of home-made aerobic activity on the level of changes in adipsin levels and visceral adiposity (VAI) in woman patients with chronic polycystic ovary syndrome in Kermanshah city.

Materials and Methods: This study was carried out among 24 women with PCOS in 2017. The samples were 12 people in the intervention group and 12 people in the control group (referred to Kermanshah health centers). The intervention group then performed home-based aerobic training for 16 weeks. Serum levels of adiposity and visceral adiposity were measured before and after the exercise program through blood sampling. SPSS V.22 software was used for data analysis and t-test with independent sample and paired t-test.

Results: There was a significant difference in the visceral adiposity index (VAI) after 16 weeks of aerobic exercise in the intervention group (P = 0.014). However, adipsin level changes were not statistically significant in the control and control groups. (P = 0.097).

Conclusion: Aerobic exercise under house supervision has a favorable effect on the visceral adiposity index in women with PCOS and can be recommended as a safe treatment for these patients.

Keywords: Polycyclic Ovarian Syndrome, Aerobic Exercise, Adipsin, Visceral Adiposity Index

1. Background

Polycystic ovary syndrome (PCOS) spread for about 10% of women’s population (1). This complication, also called the Stein Leventhal syndrome, is the most important cause of ovulation and ovulation in infertile women, which characterized by a polycyclic ovarian morphologic manifestation of pelvic ultrasonography, and at the other end of the spectrum, symptoms such as obesity, hyperandrogenism, menstrual cycle and infertility occur singly or in combination. Cold (2). The testosterone, insulin, cholesterol and triglyceride levels in these patients are higher than healthy people. However, the women with PCOS have lower levels of FSH (follicular growth stimulator), SHBG (high hormone binding globulin) and high-density lipoprotein (3-5). Adipsin tissue has been as an active hormonal system for controlling metabolism. This tissue secretes a number of proteins with biological activity (adipocytokines) (6). Adipsin is described by the CFD gene (Complement Factor D) on chromosome 10. Therefore, changes in adipsin concentration in these diseases are due to regulatory deficiencies (7). Various biological processes, including blood clotting, activation of supplements, fertility, immune system, development and repair of tissues, blood pressure, effects on body weight, absorption of nutrients, fibrin degradation, cell proliferation, bone formation and apoptosis are involved (8). Adipsin can limiting the speed of reactions in the alternate pathway. Also activates the complementary pathway of the substitution complement factor D, which is a speed limiting enzyme in the complement system as part of the intrinsic safety system. Therefore, adipsin is a major component of the immune system (9), in addition to its role in metabolism. Clinical studies have shown reduced levels of adipsin in several animal models of obesity (10, 11), while human studies have reported that increased bloodstream adiposity in metabolic diseases with body mass index (BMI) in obese, postmenopausal women.
The metabolism syndrome and obese pregnant women are related (12,13). There is increasing evidence that there is an interface loop among the complement system and inflammation, obesity, insulin resistance, and cardiovascular disease (15). There is also evidence of a link among the complement system and PCOS (14). In this regard, Chalan and his colleagues in 2016, reported a significant positive correlation among adipsin with body mass index, insulin resistance (evaluated by homeostasis model), free testosterone, C-reactive protein with high sensitivity and carotid indium media thickness. However, Hashemi et al. did not see any significant correlation among serum adipsin and polycystic ovary syndrome but reported that glucose and insulin levels were high in people with this syndrome and they had insulin resistance (15).

Considering the possibility of a direct relationship among obesity and insulin resistance and polycystic ovarian syndrome, also due to the high levels of adipsin in women with PCOS and the aim of this study was to evaluate the incidence of adipsin changes and visceral adiposity intake by a 16-week home-based aerobic exercise program in women with PCOS.

3. Materials and Methods

Current study was carried out in a semi-experimental before and after the Intervention. This study was conducted among 24 women with polycystic ovary syndrome (based on entry and exit indicators from the study (Table 1) in Kermanshah. They were divided into 2 groups (12 women) and control group (12). After obtaining a letter of consent from the women to participate in the study, women were selected to enter the study. At first, before the start of the training program, all women were evaluated for adipsin and visceral adiposity in order to register the pre-test. Then, the woman in the intervention group, in addition to prescribing drugs, performed 16 weeks of exercise protocols in a home-based aerobic exercise program. After completing the exercise protocol, adipsin and visceral adiposity indices were again measured. To examine the biochemical variables of the study (adipsin and visceral adiposity index), blood samples were taken from all subjects after at least 12 hours of fasting 24 hours before the first training session and 48 hours after the last training session (after the exercise protocol). In laboratory conditions, about 3 mL blood samples were taken from the anterior vein of the ankle. Sampling was performed at a specific time of day (8-10 am) so that adipsin levels do not change from day to day fluctuation. Blood samples were centrifuged for 15 minutes at a rate of 3000 rpm for 15 minutes and frozen at 80°C.

Serum adipsin level was determined by Sandwich ELISA using a human adipsin ELISA kit (Isotope, a Japanese student feather company) with a sensitivity of 0.39 pg/mL. Lipid indices including total blood cholesterol, HDL cholesterol and triglyceride (TG) were measured using special kits for clinical laboratory (Pars azmoon).

3.1. Evaluation of Visceral Adiposity Index

Based on the linear equation among body mass index and waist circumference, a model for the distribution of adipsin tissue (MOAD) has been proposed that has a strong correlation with visceral adiposity mass. Determined by MRI. This model was later modified for triglyceride and HDL values and the visceral adiposity index was developed as follows:

$$VAI = \left(\frac{WC}{36.58 + (1.89 \times BMI)}\right) \times \left(\frac{TG}{0.81} + \frac{1.52}{HDL}\right)$$

WC: waist circumference (cm)  
BMI: body mass index (kg/m²)  
TG: triglyceride  
HDL: high-density lipoprotein (mMol/L)

Table 1. Study Criteria

| Study Inclusion Criteria | Study Exclusion Criteria |
|--------------------------|--------------------------|
| Absence of Cushing’s syndrome and androgenic tumors, | Endocrine disorders such as hyperprolactinemia and hyperthyroidism |
| The duration of the disease for more than six months, | The use of any oral contraceptive within 30 days of pre-investigation |
| The age range of 20 to 40 years, | The use of any antidiabetic within 30 days of pre-investigation |
| The lack of participation in regular exercise in the two months before the study is | Oocyte induction or any corticosteroid substance within 30 days of pre-investigation |
| Not under the rehabilitation program and physiotherapy courses | Pregnancy |
| Not under the rehabilitation program and physiotherapy courses | Smoking, and alcohol |
| Absence of other underlying diseases: neurologist diagnosis including cardiovascular disease, epilepsy, metabolic, depression, anxiety or other psychiatric disorders, orthopedics, Convulsion and rebound disorder, severe pain in the joints of lower extremities and trunk, vestibular disease and visual impairment, any medical problems that could affect the security protocol prescribed for the patient | In case of dissatisfaction of the woman and their reluctance to continue the research process |

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3.2. Exercise

The training program was individual and on a daily basis. Its goal was to achieve specific exercise energy (ExEE) per session. During the first four weeks of the ExEE, ExEE was equivalent to four percent of the estimated individual energy needed to maintain weight during the weeks 5 through 8 to 6 percent, during the week’s 9 to 12 to 8 percent, and during the weeks 13 to 16 Rose to ten percent. The required energy was also used from the previously published equation for energy input required to maintain weight at the Pennington Research Center for people with a common life.

Energy requirement (kilocalories per day) = 1625 + 31.8 (fat free mass in kilograms) + 1.5 (fat mass in kilograms) - 187 (for females).

After calculating the Exercise Energy Expenditure (ExEE) for each session and after converting it to the amount of oxygen consumed by activity, putting this in the formula proposed by the American College of Sport Medicine for staging work (with an apparent height of 254 inches or 275 cm) and 22 steps per minute intended for the Queens College staircase test, invented by McArdell et al. In 1972) will take the time required to reach the calculated energy consumption. Came:

\[
V_{O_2} \left( \frac{mL}{kg.min} \right) = (0.2 \times f) + (1.33 \times 1.8 \times f \times h) + 3.5 \left( \frac{mL}{kg.min} \right)
\] (2)

Adipsin levels and visceral adiposity index (VAI) in the intervention group and the control group are shown in Table 3 and Figure 1. Paired t-test showed no significant difference in intra-group changes in adipsin levels in the intervention group (P = 0.097) and control group (P = 0.396). Paired t-test showed a significant difference in intra-group variation in the rate of VAI index in the intervention group before and after the intervention (P = 0.014). But this difference in the control group was not statistically significant (P = 0.097).

Based on independent t-test, there was no significant difference in the level of changes in adipsin levels among the intervention group and the control group. However, there was a significant difference between the level of VAI changes between the control group and the intervention group (Table 4).

5. Discussion

The results showed that a course of aerobic exercise training did not have a significant effect on the level of adipsin in women with polycystic ovary syndrome (PCOS). Identification of adipsin as a major cause of disease in obesity and diabetes is not long ago, and the function of this protein is not completely determined (16, 17). Xia and Cianflone reported that in adipsin levels in adipsin increased in central adipsin tissue and in adipsin tissue reduced levels of adipsin have increased with the increase in body mass index that tends toward central fat. However, in women, with the increase in body mass index, the level of adipsin is reduced, which is likely to reduce the expression of adipsin in female adipsin tissue due to limiting the development of adipsin tissue in women’s obesity (18). Hashemi et al. reported that adipocytes are lipid-secreted proteins that control the body’s metabolism and adipsin, including adipocytes, has a systemic role in lipid metabolism or other physiological systems related to the energy balance of the body, and in various studies, serum adipsin levels have been associated with a change in body mass index and insulin resistance (15). Xia and Cianflone found that in 2003, with the increase in body mass index, adipocyte levels were reduced, which is likely to reduce adipsin expression in women’s adipsin tissue by limiting the development of fatty adiposity in obesity (18). The study of Villa and Pratley showed that women with PCOS did not increase the volume of visceral fat tissue. Therefore, the distribution of fat at the abdominal points is not a complete definition for the metabolic abnormalities observed in PCOS (19). According to the results of the published studies, the lack of effect of a course of aerobic training under domestic supervision has a significant effect on...
Table 2. Descriptive Characteristics of Participants in the Intervention

| Group        | Age (Mean ± SD) | Tall (Mean ± SD) | Weight (Mean ± SD) | BMI (Mean ± SD) | P value |
|--------------|----------------|-----------------|--------------------|----------------|---------|
| Control      | 28.31 ± 4.52   | 177.22 ± 7.46   | 66.67 ± 7.39       | 20.96 ± 2.09   | 0.495   |
| Intervention | 31.64 ± 4.28   | 174.38 ± 5.19   | 64.17 ± 5.67       | 21.71 ± 1.76   | 0.716   |

Table 3. Mean and Standard Deviation of Adipsin and Visceral Adiposity Index (VAI) in the Intervention Group and Control Group

| Group        | Pre-Test (Mean ± SD) | Post-Test (Mean ± SD) | P Value |
|--------------|----------------------|-----------------------|---------|
| Control      | Adipsin (ng/dL)      | 15.22 ± 3.68          | 14.98 ± 4.17 | 0.369 |
|              | VAI                  | 36.43 ± 7.68          | 35.96 ± 7.46 | 0.791 |
| Intervention | Adipsin (ng/dL)      | 16.4 ± 3.27           | 15.97 ± 4.65 | 0.097 |
|              | VAI                  | 35.12 ± 7.46          | 29.86 ± 8.64 | 0.014* |

Table 4. Comparison of Mean Changes in Adipsin and VAI Levels Among Groups in the Intervention Group and Control Group

| Variable | Control | Intervention | P Value |
|----------|---------|--------------|---------|
| Adipsin  | Pre-test| 15.22 ± 3.68 | 16.4 ± 3.27 | 0.740 |
|          | Post-test| 14.98 ± 4.17 | 15.97 ± 4.65 | 0.631 |
| VAI      | Pre-test| 36.43 ± 7.68 | 35.12 ± 7.46 | 0.869 |
|          | Post-test| 35.96 ± 7.46 | 29.86 ± 8.64 | 0.036* |

The results of this study showed that there was a significant difference in VAI in women with polycystic ovary syndrome after intensive intramural training, but VAI had no significant difference before and after the control in the control group, therefore, VAI levels could be improved in the study group was attributed to the effect of severe intolerance training. Earlier, some studies have reported the reduction of central and subcutaneous and visceral fat intake after a physical course (20). Yip et al. reported a 31 percent reduction in visceral fat and a 26 percent reduction in abdominal subcutaneous fat following a loss weight ith diet (21). Takami et al. also observed a 25.8% reduction in fat and 17.2% lower abdominal subcutaneous fat after an aerobic training period (20). Which is consistent with the results of this study, Exercise may reduce the size of the chamber without changes in body mass index (22). In addition, exercise, even without weight loss, reduces visceral adiposity and prevents obesity (22). Irwin et al. also found that about 200 minutes of exercise per week despite a modest decrease in weight resulted in a significant reduction in visceral adiposity in postmenopausal women who were overweight and also overweight, 4.2% of total body fat And 6.9% of the visceral fat of these individuals has been reduced without limiting calorie intakes (23). Mora et al., In a study entitled with New and Traditional Physical Activity and Body Mass Index Cardiovascular Indicators in Women, concluded that lower levels of physical activity and increased body mass index (BMI) were independently associated with increased total cholesterol, Triglyceride and inflammatory markers such as CRP (24). The increase in body mass index increases the risk of cardiovascular disease by 8% per unit and, on the contrary,
with the increase in physical activity, the risk of cardiovascular disease decreases (24). Programs that increase the capacity of skeletal muscle to use fats may play an important role in controlling people’s weight and reducing cardiovascular risk factors (23). In this regard, the amount of weight loss and body mass index and fat percentage are related to the amount of calories and the amount of energy expenditure. It has also been shown that during the aerobic training of the endocrine system, by increasing the levels of epinephrine, norepinephrine, growth hormone and cortisol, the lipid oxidation increases, and by increasing the callus and use of free fatty acids, energy needs are provided, thereby causing reduces body fat (20). Improving body composition following intense periodic exercises may be due to increased ability to buffer hydrogen ions. It is also likely that the increase in VO max exercises may be due to increased ability to buffer hydrogen ions.

5.1. Conclusion

The results of this study showed that performing 16 weeks of homeopathic aerobic exercise can improve and decrease the visceral fat index in women with PCOS (Polycystic Ovary Syndrome). But there is no effect on the level of adipin. 16 weeks of aerobic training with decreasing body mass index, fat mass and total cholesterol index have a significant effect on the visceral fat index and has a significant effect on the improvement of symptoms of polycystic ovary syndrome.

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Footnotes

Conflict of Interests: The authors declare no conflict of interest.

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References

1. Nazari T, Biat R. [Impact of metformin in girls with polycystic ovary syndrome]. Prolific and Non Prolific J. 2005;16:45–9. Persian.

2. Amer SA. Polycystic ovarian syndrome: diagnosis and management of related infertility. Obstet Gynecol Rep Med. 2009;19(10):263–70. doi: 10.1016/j.ogrm.2009.06.006.

3. Hertle E, van Greevenbroek MM, Stenhouwer CD. Complement C3: an emerging risk factor in cardiometabolic disease. Diabetologia. 2012;55(4):881–4. doi: 10.1007/s00125-012-2462-z. [PubMed: 2228263]. [PubMed Central: PMC3293998].

4. Pasquali R, Casimirri F, Balestra V, Flamia R, Melchionda N, Fabbri R, et al. The relative contribution of androgens and insulin in determining abdominal body fat distribution in premenopausal women. J Endocrinol Invest. 1999;14(10):839–46. doi: 10.1007/BF03347939. [PubMed: 1802922].

5. Peiris AN, Sothmann MS, Alman E, Kissebah AH. The relationship of insulin to sex hormone-binding globulin: role of adiposity. Fertil Steril. 1989;52(1):69–72. doi: 10.1016/s0015-0282(16)60791-4.

6. Spiegelman BM, Flier JS. Obesity and the Regulation of Energy Balance. Cell. 2001;100(4):531–43. doi: 10.1016/s0092-8674(01)00249-9.

7. Platt KA, Min HY, Ross SR, Spiegelman BM. Obesity-linked regulation of the adipin gene promoter in transgenic mice. Proc Natl Acad Sci U S A. 1999;96(19):2798420. doi: 10.1073/pnas.96.19.2798420. [PubMed Central: PMC298090].

8. Fruhbeck G, Gomez-Ambrosi J, Muruzabal FJ, Burrell MA. The adipocyte: a model for integration of endocrine and metabolic signaling in energy metabolism regulation. Am J Physiol Endocrinol Metab. 2001;280(6):E827–47. doi: 10.1152/ajpendo.2001.280.6.E827. [PubMed: 1150765].

9. White RT, Damm D, Hancock N, Rosen BS, Lowell BB, Usher P, et al. Human adipin is identical to complement factor D and is expressed at high levels in adipose tissue. Journal of Biological Chemistry. 1992;267(1):920–3.

10. Pomeroy C, Mitchell J, Eckert E, Raymond N, Crosby R, Dalmasso AP. Effect of body weight and caloric restriction on serum complement proteins, including Factor D/adipin: studies in anorexia nervosa and obesity. Clin Exp Immunol. 1997;108(3):507–15. doi: 10.1046/j.1365-2249.1997.392287.x. [PubMed: 9102906]. [PubMed Central: PMC904692].

Int J Health Life Sci. 2020; 6(2):e97400.

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11. Cook KS, Min HY, Johnson D, Chaplinsky RJ, Flier JS, Hunt CR, et al. Adipsin: a circulating serine protease homolog secreted by adipose tissue and sciatic nerve. Science. 1987;237(4813):402–5. doi: 10.1126/science.3299705. [PubMed: 3299705].

12. Chedraui P, Perez-Lopez FR, Escobar GS, Palla G, Monnt-Guevara M, Cechi E, et al. Circulating leptin, resistin, adiponectin, visfatin, adipsin and ghrelin levels and insulin resistance in postmenopausal women with and without the metabolic syndrome. Maturitas. 2014;79(1):86–90. doi: 10.1016/j.maturitas.2014.06.008. [PubMed: 25015014].

13. Sivakumar K, Bari MF, Adaikalakoteswari A, Guller S, Weickert MO, Randeva HS, et al. Elevated fetal adipsin/acylation-stimulating protein (ASP) in obese pregnancy: novel placental secretion via Hofbauer cells. J Clin Endocrinol Metab. 2013;98(10):4113–22. doi: 10.1210/jc.2012-4293. [PubMed: 23956345]. [PubMed Central: PMC3790615].

14. Oktenli C, Ozgurtas T, Dede M, Sanisoglu YS, Yenen MC, Yesilova Z, et al. Metformin decreases circulating acylation-stimulating protein levels in polycystic ovary syndrome. Gynecol Endocrinol. 2007;23(12):710–5. doi: 10.1080/09513590701666571. [PubMed: 18075846].

15. Hashemi F, Poudeh Q, Ramezani Tehrani F, Hedayati M; Yaghmaei. The relationship between serum levels of Adipsin with polycystic ovary syndrome (PCOS). Razi Journal of Medical Sciences. 2012;19(99).

16. Clark AM, Thornley B, Tomlinson L, Galletley C, Norman RJ. Weight loss in obese infertile women results in improvement in reproductive outcome for all forms of fertility treatment. Hum Reprod. 1998;13(6):502–5. doi: 10.1093/humrep/dej150. [PubMed: 9688382].

17. Cianflone K, Roncari DA, Maslowska M, Baldo A, Forden J, Snieder AD. Adipsin/acylation stimulating protein system in human adipocytes: regulation of triglyceride synthesis. Biochemistry. 1994;33(32):9489–95. doi: 10.1021/bi00098a004. [PubMed: 8068621].

18. Xia Z, Cianflone K. Acylation-stimulating protein precursor proteins in adipose tissue in human obesity. Metabolism. 2003;52(10):1360–6. doi: 10.1016/s0026-0495(03)00254-3.

19. Villa J, Pratley RE. Adipose tissue dysfunction in polycystic ovary syndrome. Curr Diab Rep. 2011;11(3):279–84. doi: 10.1007/s11892-011-0189-8. [PubMed: 21424395].

20. Takami K, Takeda N, Nakashima K, Takami R, Hayashi M, Ozeki S, et al. Effects of dietary treatment alone or diet with voglibose or glyburide on abdominal adipose tissue and metabolic abnormalities in patients with newly diagnosed type 2 diabetes. Diabetes Care. 2002;25(4):658–62. doi: 10.2337/diacare.25.4.658. [PubMed: 1199121].

21. Yip I, Go VL, Hershman JM, Wang HJ, Elshoff R, DeShields S, et al. Insulin-leptin-visceral fat relation during weight loss. Pancreas. 2001;23(2):197–203. doi: 10.1097/00006676-200108000-00010. [PubMed: 11484922].

22. Freedland ES. Role of a critical visceral adipose tissue threshold (CVATT) in metabolic syndrome: implications for controlling dietary carbohydrates: a review. Nutrition & metabolism. 2004;1(1):32.

23. Irwin ML, Yasui Y, Ulrich CM, Bowen D, Rudolph RE, Schwartz RS, et al. Effect of exercise on total and intra-abdominal body fat in postmenopausal women: a randomized controlled trial. JAMA. 2001;289(3):323–30. doi: 10.1001/jama.289.3.323. [PubMed: 12525231].

24. Mora S, Lee IM, Buring JE, Ridker PM. Association of physical activity and body mass index with novel and traditional cardiovascular biomarkers in women. JAMA. 2006;295(12):1412–9. doi: 10.1001/jama.295.12.1412. [PubMed: 16550771].

25. Duclos M, Oppert JM, Verges B, Coliche V, Gautier JF, Guzennece Y, et al. Physical activity and type 2 diabetes. Recommendations of the SFD (Francophone Diabetes Society) diabetes and physical activity working group. Diabetes Metab. 2013;39(3):205–16. doi: 10.1006/j.dibet.2013.01.005. [PubMed: 23643351].

26. Mann S, Beedie C, Balducci S, Zanuso S, Allgrove J, Bertiau F, et al. Changes in insulin sensitivity in response to different modalities of exercise: a review of the evidence. Diabetes Metab Res Rev. 2014;30(4):257–68. doi: 10.1002/dmrr.2488. [PubMed: 24130081].