

# Status of serum AMH and lipid profile in polycystic ovarian syndrome

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## Abstract

Polycystic ovarian syndrome (PCOS) is the most common causes of chronic anovulation in young women and affects 5 to 10 % of the female population. Anti-Mullerian Hormone (AMH) level indicate the quantity of the ovarian follicle pool and may be a useful marker of ovarian reserves. Serum AMH level can determine the severity of PCOS women and its comparison with control subject the present study was planned. Study was carried out in M.G.M. medical college, Govt. Holkar science college and K.R.G's Blessed mom centre from June -2016 to December-2017. The study population consisted of 60 subject among them 30 cases suffering from PCOS aged between 20 to 40 year and 30 age matched healthy women as control. Fasting blood sample were collected from each subject and analyzed for AMH level and Lipid profile by ELISA method and enzymatic method on fully automated biochemistry analyzer. Results Revealed that significant increased serum AMH, Cholesterol, triglycerides, LDL, VLDL levels and decreased HDL level were observed in PCOS cases when compare to control subjects. Study concluded elevated AMH level leads to increases risk of Polycystic Ovarian Syndrome, Hyperlipidemia and associated complications

**Key Word:** AMH- Anti-Mullerian Hormone, PCOS- Polycystic ovarian syndrome, LDL-Low Density Lipoprotein, HDL-High Density Lipoprotein, VLDL-Very Low Density Lipoprotein, FSH-follicle Stimulating Hormone

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## INTRODUCTION

Polycystic Ovarian Syndrome(PCOS)is the one of the most common heterogeneous endocrine disorders in women of reproductive age affecting the reproductive, endocrine and metabolic functions<sup>1,2</sup> and its association with menstrual dysfunction and subfertility<sup>3</sup>.Prevalence of PCOS is around 5 to 10 % of reproductive age female<sup>4</sup>.Anti-MullerianHormone(AMH),also known as Mullerian Inhibiting Substance(MIS), is a homodimeric glycoprotein(5). The AMH belongs to the Transforming Growth Factor beta super family. The gene encoding AMH is located in the short arm of chromosome 19<sup>6</sup>.

AMH was produced from 36 weeks of gestation in human granulosa cells and was expressed until menopause<sup>7</sup>. AMH protein expression begins at the primary follicle stage, declines and absent in follicles larger than 8 mm<sup>8</sup>. Important role for AMH in folliculogenesis,<sup>9</sup>.During folliculogenesis, AMH play an inhibitory effect on the primordial follicles recruitment as well as on the responsiveness of growing follicles to FSH, suppressing the FSH depending aromatase and also, diminish the LH receptors, thus helping the selection of the dominant follicle or role in follicle recruitment<sup>10</sup> Cessation of AMH production essential for dominant follicle selection<sup>11</sup>.The distinctive feature of PCOS is failure of follicular maturation, despite initial recruitment resulting in anovulation and accumulation of preantral and small antralfollicles, which contribute high production of AMH<sup>12,13,14</sup>. Increase AMH may contribute to the development of hyperandrogenism in women with PCOS, and show positive correlation with androgen<sup>15,16</sup>. Hyperandrogenism accelerates pre antral and antral follicular growth in the ovary and increased LH result in premature luteinizationcauring follicular arrest<sup>17</sup>, driving increase AMH levels. AMH levels may be related to the severity of PCOS<sup>18</sup>, higher concentration of AMH in

women with PCOS features including polycystic ovaries (PCO), anovulation, hyperandrogenism and Insulin Resistance<sup>19</sup>. AMH and Insulin resistance in PCOS may reflect indirect regulation through androgen<sup>20</sup>. Hyperlipidemia is one of the main metabolic characteristics of PCOS patients. Insulin Resistance is considered to play a role in defected lipid profile about 70% of women with PCOS have at least one abnormal lipid constituent<sup>21</sup>. Hyperandrogenism also contributes for alerted lipid profile<sup>22</sup>, with elevated total cholesterol, Triglycerides and low density lipoprotein (LDL) and low levels of high density lipoproteins (HDL)<sup>7</sup>. In the study the AMH level deranged Lipid metabolism and other PCOS related complications in women suffering from polycystic ovarian syndrome and its correlation with healthy control subject.

**MATERIAL AND METHODS**

This study was carried out in the M.G.M. Medical college, Government Holker Science College and K. R. G's Blessed Mom Centre, Indore (M.P.), during June 2016 to December 2017. Study comprised total 60 Subjects divided in to two groups control and cases. 30 Healthy women aged between 20 to 40 year taken as control and 30 PCOS patients aged between 20 to 40 year taken as cases. Fasting blood sample from each cases and control were collected and analyze for AMH, Cholesterol, Triglycerides, HDL, LDL, VLDL levels. Serum AMH level were measured using Enzyme Linked Immunosorbent Assay (ELISA). Cholesterol, triglycerides, HDL, LDL, VLDL, were measured by enzymatic method of fully automated biochemistry analyzer. Data were analyzed by using Statistical Program for Social Sciences Version (SPSS) software and data were expressed as mean and standard deviation. (Mean+SD) Comparison done by using student t-test and p-value. P-value <0.05 taken as significant and p value < 0.001 taken as highly significant

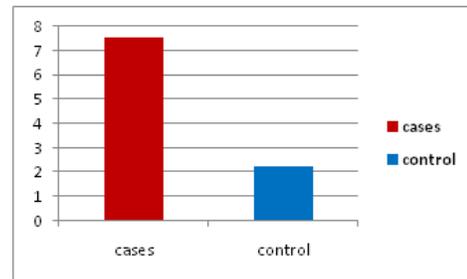
**OBSERVATION**

**Table 1:** Comparison of Serum AMH level cases and control

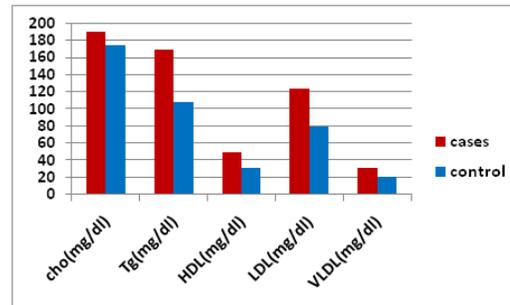
Parameters	control(n=60)	PCOS (n=60)	p-value
AMH (ng/ml)	2.22 ± 1.12	7.54 ± 2.11	<0.001

**Table 2:** Comparison of lipid profile levels in cases and control

Parameter	Control(n=60) Mean±SD	Cases(n=60) Mean±SD	P value
CHO (mg/dl)	174±21	197±19	<0.001
TG (mg/dl)	108±25.87	169±18.45	<0.001
HDL (mg/dl)	50.13±12	31±6.14	<0.001
LDL (mg/dl)	79± 30	123±22	<0.001
VLDL(mg/dl)	21±6.99	31±5.40	<0.001



**Figure 1:** Comparison of serum AMH level between cases and control



**Figure 2:** Comparison of lipid profile levels in cases and control

**RESULTS**

Result revealed that the mean serum AMH level in cases was 7.54± 2.11 and mean serum AMH level in control subject was 2.22± 1.12. Result showed highly Significant(0.001) increase in serum AMH level in cases when compare to control There was statistically significant(0.001) increased levels of total cholesterol, Triglyceride, LDL, VLDL in PCOS group when compared to the controle group (0.001), with significant decreased HDL level in PCOS group when compared to control.

## DISCUSSION

The study show AMH level are 2 to 3 fold higher in women with PCOS compared with healthy women. The role of increased AMH in the follicular arrest in PCOS by inhibiting FSH early in folliculogenesis<sup>23</sup>. Elevated AMH contributes to the pathogenesis of PCOS, factor which are closely related to PCOS pathophysiology such as increased androgen level and insulin resistance may be implicated AMH level<sup>16</sup>. PCOS is characterized by an increase number of follicles at all growing stages<sup>24</sup>, this increase is particularly seen in the pre-antral and small antral follicles, those which primarily produce AMH<sup>9</sup>. Increase serum AMH levels in PCOS would also reflect an intrinsic dysregulation of the granulosa cells, in which AMH, itself could be involved since an over expression of the AMH receptor type 2 (AMHR 2) has also been demonstrated<sup>25</sup>. The expression patterns of AMH and its type two receptor (AMHR2) in the postnatal ovary indicated the importance of AMH signalling in ovarian folliculogenesis<sup>10</sup>. Granulosa cells of polycystic ovaries have increased AMH mRNA expression<sup>25</sup>, it is not only the increase number of follicles, with resultant increase granulosa cell mass, but also greater production by individual granulosa cells that is underlying AMH over production in PCOS<sup>23</sup>. Increased AMH may contribute to the development of hyperandrogenism in PCOS women which may be mediated by the reduction in aromatase activity in granulosa cells of polycystic ovaries<sup>7,11</sup>, Aromatase convert androgen to estrogens in granulosa cells.<sup>26</sup> Increased androgen can increase AMH through augmenting follicular growth and Hyperandrogenism, clinically manifested by hirsutism, acene, follicular arrest and anovulation form cyst and cause a polycystic ovaries<sup>27,28</sup>. The Role of androgen, such as dehydroepiandrosterone (DHEA) and DHEA-S, Promoting insulin resistance in PCOS women<sup>29</sup>. Lipid abnormalities were closely related to Insulin Resistance, obesity in PCOS, contribute partly through lipolysis stimulation and altered expression of lipoprotein lipase and hepatic lipase<sup>30</sup>. Increase in triglycerides may be due the accumulation of triglycerides, due to the increase lipogenesis, due to reduced oxidation of fatty acids increased secretion of VLDL particles by the liver results elevated plasma triglycerides concentration. This may occur due to insulin resistance also contributes more catabolism of HDL-C particle and formation of LDL-C<sup>21</sup>. Hyperandrogenism also contribute for alerted lipid profile and associated with increased hepatic lipase activity has role in catabolism of HDL-C particles<sup>31</sup>. Change of lipid lead to increased risk for premature atherosclerosis.<sup>21</sup>. LDL particles are atherogenic and are strongly associated with coronary artery disease.<sup>32</sup>

## CONCLUSION

Elevated levels of AMH appear to play an important role in long term disruption of ovarian physiology and contributes the pathogenesis of PCOS. increased AMH direct interaction with androgen and indirect role of Insulin Resistance in Pathophysiology of reproductive and metabolic dysfunction in PCOS and severity of PCOS Including, Polycystic Ovaries (PCO), anovulation, hyperandrogenism and Insulin Resistance, dyslipidemia, hypertension, obesity, infertility, Premature atherosclerosis, Type 2 diabetes, cardiovascular disease and long term complication. Early estimation of AMH level in women may decrease the risk of PCOS and associated complications.

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