

Evaluation of the role of Fetuin A in pathophysiology of polycystic ovarian syndrome in adolescents

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Introduction and Objective

Polycystic ovarian syndrome (PCOS); is an endocrinopathy characterised by overlap of hyperandrogenism and hyperinsulinism and chronic anovulation in which both genetic and environmental factors play a role. Etiopathogenesis is still not clearly defined. Fetuin-A is a serum glycoprotein. The dominant source is liver. It is shown to play regulatory functions in many inflammatory processes. Here we aim to define the relationship of fetuin-A levels with hyperandrogenism and hyperinsulinism in PCOS patients and study the role in pathophysiology.

Method

Adolescent patients with similar age and BMI and diagnosed as PCOS according to 'Endocrine Society Clinical Practice' guides, were included in the study. Subgroups of PCOS and control group were defined as obese and non-obese according to the BMI being above or below VKİ 25 kg/m². Levels of LH, FSH, total & free testosterone, SHBG, 1-4delta androstenedione, and DHEAS results were recorded in PCOS patients, Free androgen index was calculated. Fasting glucose levels, insulin, lipid profile, SGOT, SGPT were recorded in control patients (PCOS and obese) HOMA-IR was calculated. Cut off value for HOMA-IR was $\geq 3,82$ (3). All patients had blood drawing for fetuin-A and hsCRP. Ovarian size was measured by ultrasonography.

Results

Mean age was 16,4 \pm 1,1 years in 38 patients with PCOS and 16,2 \pm 0,98 years in control group (n=40), mean BMI of PCOS group was 27,9 \pm 6 kg/m² and 27,2 \pm 6,7 kg/m² in control group and there was no significant difference. **Mean Fetuin-A level in PCOS group was significantly higher than the control group (p:0,035). (PCOS 583,1 \pm 197,45, control 492,08 \pm 176,3 ng/ml).**

When Obese-PCOS and Non-Obese PCOS groups were compared;

Fetuin-A, FSH, LH, total and free testosterone, 1,4 Δ AS, DHEAS and SAI levels were similar. Obese-PCOS patients had significantly **lower SHBG and HDL levels (p=0,012/p:0,017)** and **higher cholesterol, LDL, triglycerid, HOMA-IR and HSCRP levels when compared to non-obese PCOS patients (p=0,014-0,031-0,006-0,015-0,024).**

Non-obese PCOS and Non-obese control groups were compared;

Fetuin-A, hHsCRP and all other parameters were similar

When obese PCOS and Obese control groups were compared;

In obese-PCOS group, **Fetuin-A levels were significantly higher than obese-control group (p:0,016)**. Cholesterol, LDL, HDL, HOMA-IR and HSCRP levels and all other parameters were similar.

Obese-PCOS and Non-obese healthy control groups were compared;

BMI, BMI-SDS, HSCRP and Fetuin-A were significantly higher in obese PCOS patients (0,001-0,001-0,035, 0,013).

Highest fetuin-A level was detected in obese-PCOS group. This was significantly different from obese-control and non-obese control groups (p:0,016, 0,013). Also in PCOS group fetuin-A levels was positively correlated with , triglycerid (r:0,470, p:0,003), LH (r:0,416, p:0,009), LH/FSH ratio (r:0,381, p:0,018), total testosterone (r:0,313, p:0,056), 1,4 Δ AS (r:0,441, p:0,008) and SAI (r:0,425, p:0,05).

Table1: Clinical and laboratory properties of PCOS group and control group

Group	PCOS with obesity (n:22)	Obesity control (n:21)	P	Non-obese PCOS (n:16)	Non-obese control (n:19)	P
Age (years)	16,5 \pm 1,6	16,4 \pm 0,9	0,85	16,3 \pm 1,4	16,2 \pm 1	0,98
Height SDS	-0,34 \pm 1	0,28 \pm 1,5	0,25	-0,47 \pm 0,8	-0,16 \pm 0,7	0,21
BMI (kg/m ²)	31,5 \pm 4,2	33 \pm 3,8	0,17	22,07 \pm 2,8	21,1 \pm 2,08	0,097
BMI SDS	2,65 \pm 0,64	2,9 \pm 0,6	0,1	0,45 \pm 0,9	0 \pm 0,89	0,07
hsCRP (mg/dl)	0,31 (0,03-0,5)	0,29 (0-0,5)	0,78	0,15 (0,03-0,5)	0,22 (0,05-0,5)	0,57
Fetuin A (ng/ml)	618 (286-927) 648,8\pm175,2	494,2(255-1112) 479,5\pm255,9	0,016	535,2(287-945) 573,3\pm221,2	417,7 (210-923) 448\pm144,2	0,054

Table2: Fetuin-a correlation in pcos group

Fetuin A	r	p
TG	0,470	0,003
LH	0,416	0,009
LH/FSH	0,381	0,018
deltaAS	0,441	0,008
T. testosteron	0,313	0,056
FAI	0,325	0,05

Conclusion

Fetuin A levels in our PCOS patients were significantly higher than the control group. In obese PCOS patients, fetuin-A levels were slightly higher than the non-obese PCOS patients and significantly higher than patients in obese control group with similar BMI, lipid profile and HOMA-IR levels. These results put forward the relationship of androgens with Fetuin-A and can direct further studies

References

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