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May AMH levels distinguish LOCAH from PCOS among hirsute women?

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ABSTRACT

Objective: To determine whether women with polycystic ovary syndrome (PCOS) would be distinguishable from women with late onset congenital adrenal hyperplasia (LOCAH) on the basis of antimullerian hormone (AMH) levels.
Study design: PCOS was diagnosed in 170 women; 105 were polycystic ovary morphology (PCOM)+/oligo-anovulation (OA)+/hyperandrogenism (HA)+, 40 PCOM+/OA−/HA+ and 25 PCOM−/OA+/HA+. These three groups were compared with 25 women in whom LOCAH was diagnosed.
Results: The mean serum AMH levels were 8.12 ± 1.85 ng/ml in PCOM+/OA+/HA+ group, 5.34 ± 1.82 ng/ml in PCOM+/OA−/HA+ group, 3.02 ± 1.76 ng/ml in PCOM−/OA+/HA+ group and 4.43 ± 1.29 ng/ml in LOCAH group. The mean AMH level in PCOM+/OA+/HA+ group was approximately twofold higher than the mean AMH level measured in LOCAH group ($p < 0.001$). Women with PCOM+/OA−/HA+ had higher serum AMH levels than those with LOCAH, women with LOCAH had higher serum AMH levels than those with PCOM−/OA+/HA+ but these differences were not statistically significant ($p > 0.05$).
Conclusions: AMH is not suitable for distinguishing LOCAH from all types of hyperandrogenic patterns of PCOS, but is only applicable for a specific subtype, such as PCOS patients with three main diagnostic criteria. Therefore, ACTH stimulation test remains an essential clinical tool to diagnose LOCAH.

Introduction

Androgen excess is one of the most common endocrine disorders of women at reproductive age and occurs in approximately 10% of this population [1]. Clinical manifestations of hyperandrogenism include hirsutism, acne and/or seborrhea, androgenic alopecia, menstrual irregularities and rarely when prolonged, virilization and masculinization [1].

The most common cause of androgen excess is the polycystic ovary syndrome (PCOS), with late onset congenital adrenal hyperplasia (LOCAH), the hyperandrogenic insulin-resistant acanthosis nigricans syndrome and ovarian or adrenal androgen-secreting tumors occurring less frequently [2]. Distinguishing between PCOS and LOCAH clinically is very difficult.

LOCAH is an autosomal recessive adrenocortical disorder that occurs in approximately 1 in 1000 and is characterized by partial deficiency of steroidogenic enzymes essential for cortisol biosynthesis [3]. LOCAH is most commonly due to an enzyme deficiency of 21-hydroxylase, 3β-hydroxysteroid dehydrogenase or 11β-hydroxylase [4]. The diagnosis of LOCAH is based on noticeably supranormal basal or ACTH-stimulated 17-hydroxyprogesterone (17-OHP) levels [5]. Clinical manifestations of LOCAH include hirsutism, acne, alopecia, and ovulatory and menstrual dysfunction. The incidence of LOCAH varies geographically, presumably because of ethnic and racial clustering [6].

PCOS is one of the most common endocrine abnormalities in women of reproductive-age characterized by ovulatory dysfunction and hyperandrogenism, affecting 5–10% of women in this age-group [7]. PCOS diagnosis is based mainly on clinical and physical findings. PCOS is defined as the presence of any two of the following three criteria: (1) polycystic ovary morphology (PCOM) on ultrasound scan; (2) oligo-ovulation and/or anovulation (OA); and (3) clinical or biochemical evidence of androgen excess (HA); on condition that other etiologies (congenital adrenal hyperplasia, androgen-secreting tumors, Cushing's syndrome) have been excluded [8]. As with LOCAH, clinical manifestations of this syndrome may include dermatologic finding of hyperandrogenism and ovulatory and menstrual dysfunction. Antimullerian hormone

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(AMH) level indicates the quantity of the ovarian follicle pool [9] and is an important marker of PCOS [10]. Pigny et al. showed that women with PCOS have 2- to 3-fold higher levels of AMH than healthy women [11]. This is due to the increase in the number of follicles in PCOS patients, as well as the increase in AMH production per follicle. Overall, it is difficult to distinguish PCOS clinically from LOCAH. As patients with PCOS have significantly higher serum AMH levels than control subjects, we hypothesized that AMH levels may distinguish LOCAH from PCOS among hirsute women.

The aim of this study was to determine whether women with PCOS would be distinguishable from women with LOCAH on the basis of AMH levels.

Materials and methods

Between December 2009 and January 2012, Caucasian women, who had acne, hirsutism and menstrual dysfunction (*oligomenorrhea* and/or *amenorrhea*) suspected to be due to hyperandrogenism with recorded AMH concentrations were retrospectively identified. The inclusion criteria were as follows: <40 years of age, presence of LOCAH or the hyperandrogenic pattern of PCOS, no previous history of ovarian surgery, no thyroid or prolactin hormone level abnormalities and no hormonal therapy in the 6 months before entering the study. Following these criteria, the final study population consisted of 195 consecutive patients. PCOS was diagnosed in 170 women; 105 were PCOM+/OA+/HA+, 40 were PCOM+/OA−/HA+ and 25 were PCOM−/OA+/HA+. These three groups were compared with 25 women in whom LOCAH was diagnosed. An approval from the Human Ethics Committee of Istanbul University was obtained.

Blood sampling was performed in the early follicular phase (between cycle day 3 and 5). In oligo- or amenorrheic patients, the last menstrual period was either spontaneous or induced by the administration of progesterone.

Diagnosis of PCOS was established according to the Rotterdam criteria with the presence of at least two of the following diagnostic features after the exclusion of other etiologies: oligo- or anovulation, clinical and/or biochemical signs of hyperandrogenism and polycystic ovaries shown by ultrasonography [8]. PCOM was defined as the presence of 12 or more 2- to 9-mm-diameter follicles in each ovary or an increased ovarian volume of >10 ml. The ovaries were localized and scanned. OA was defined as the presence of oligomenorrhea (menstrual cycle of >35 days) or amenorrhea (i.e., no menstrual bleeding during the last 3 months). HA was defined as the presence of elevated serum androgen levels or the presence of hirsutism as defined by a modified Ferriman–Gallwey hirsutism score ≥8 [12]. Nine areas of the body and face were assessed for hair growth on a scale of 0–4: upper lip, chin, arms, chest, upper and lower abdomen, thighs, and upper and lower back. The total modified Ferriman–Gallwey score represents the sum of all of the scores. The presence of comedones on the face, neck, upper chest, upper back and upper arms were classified as acne [13]. The presence of greasy or oily and shiny skin on the nasolabial folds, the forehead or behind the ear and the hair were defined as seborrhea [14]. Hair loss was defined according to Ludwig classification system [15]. Body mass index (BMI) was calculated with the following formula: weight (kg)/height $(m)^2$.

The screening test for LOCAH can usually be carried out by measuring serum 17-OHP levels in the morning during early follicular phase. The patients who had 17-OHP levels higher than 2 ng/ml and hyperandrogenemic symptoms were accepted patients with elevated levels of 17-OHP and we performed ACTH stimulation test on these patients. None of the women received any hormonal medication for the last 6 months before the test. ACTH stimulation test was performed on all women in the fasting state and in the supine position between 08.00 and 09.00 am on day 3–5 of their cycle. After baseline blood was sampled, 0.25 mg synthetic ACTH (Synachten, Ciba-Geigy, Basel, Switzerland) was injected intravenously and blood samples were obtained after 1 h. Both the 0- and 60-min samples were assayed for 17-OHP levels. An ACTH-stimulated 17-OHP level >10.0 ng/ml was considered as the criterion of LOCAH [4].

All blood samples for AMH measurement were collected in a lithium-heparin tube. AMH concentrations were measured with an enzymatically amplified two-sided immunoassay (DSL-10-14400 Active Mullerian Inhibiting Substance/AMH enzymelinked immunosorbent assay [ELISA] kit, Diagnostic Systems Laboratories [DSL], Webster, TX). The theoretical sensitivity of the method was 0.006 ng/ml, the intraassay coefficient of variation for high values was 3.3%, and the inter-assay coefficient of variation for high values was 6.7%. Serum levels of Estradiol (E2), follicle-stimulating hormone (FSH), luteinizing hormone (LH) were measured using an Immulite 2000 (Diagnostic Products Corp., Los Angeles, CA). Serum levels of 17-OHP were assayed in competitive immunoenzymatic colorimetric method (DiaMetra S.r.I. Headquater, Via Garibaldi, 18-20090, Segrate, Milano, Italy). The sensitivity of the assay was 0.009 ng/ml. The intra- and inter-assay coefficient of variations were <7.4% and <13%, respectively. Reference values are between 0.2 and 1.3 ng/ml in follicular phase in women. Serum levels of total testosterone (total T), free testosterone (free T), dehydroepiandrosterone-sulfate (DHEA-S) were assayed in competitive immunoenzymatic colorimetric method (DiaMetra S.r.I. Headquater, Via Garibaldi, 18-20090, Segrate, Milano, Italy).

For sonographic imaging, we used a 6.5-MHz vaginal transducer (model EUB-415; Hitachi Medical Corp., Tokyo, Japan).

Statistical analysis

Statistical analysis was performed using SPSS 17.0 for Windows. Data are presented as mean ± SD. Data were checked for normality before analysis with the Kolmogorov–Smirnov test, and nonnormally distributed data (AMH, LH, E2, 17-OHP, total T, free T) were transformed logarithmically. Group means were compared using the ANOVA test. Distributions of categorical data were evaluated using x^2 tests. Receiver operating characteristic (ROC) curves of AMH and 17-OHP levels were constructed to examine the diagnostic test performance for the groups. An area under the curve (AUC) value of 0.5 indicated that the test had no discriminative power, and a value of 1.0 indicated that the test had perfect discrimination. Differences among groups were considered to be significant if the p value was <0.05.

Results

Of the 195 subjects who participated in this study, 105 (53.8%) had PCOM+/OA+/HA+, 40 (20.6%) had PCOM+/OA−/HA+, 25 (12.8%) had PCOM−/OA+/HA+ and 25 (12.8%) had LOCAH. Data obtained from the four groups are shown in Table 1. No statistically significant differences were found among the four groups in terms of BMI. The mean age of the PCOM+/OA+/HA+ and PCOM+/OA−/HA+ groups were higher than LOCAH group.

The mean serum AMH levels were 8.12 ± 1.85 ng/ml in PCOM+/OA+/HA+ group, 5.34 ± 1.82 ng/ml in PCOM+/OA−/HA+ group, 3.02 ± 1.76 ng/ml in PCOM−/OA+/HA+ group and 4.43 ± 1.29 ng/ml in LOCAH group. The highest AMH levels were found in PCOM+/OA+/HA+ group. The mean AMH level in PCOM+/OA+/HA+ group was approximately two fold higher than the mean AMH level measured in LOCAH group ($p < 0.001$).

Fig. 1 presents the mean serum AMH levels of the four groups. Women with PCOM+/OA−/HA+ had higher serum AMH levels than those with LOCAH and women with LOCAH had higher serum AMH

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Table 1
Biochemical and demographic data of subjects.

	PCOM+/OA+/HA+	PCOM+/OA−/HA+	PCOM−/OA+/HA+	LOCAH
Patient number (%)	105 (53.8%)	40 (20.6%)	25 (12.8%)	25 (12.8%)
Age (years)	24.8 ± 5.0[c]	25.0 ± 4.7[c]	23.1 ± 5.8	21.5 ± 3.9
BMI (kg/m^2)	25.8 ± 5.2	24.3 ± 4.1	27.5 ± 6.2	25.2 ± 4.3
AMH (ng/ml)	8.12 ± 1.85[a,b,c]	5.34 ± 1.82[b]	3.02 ± 1.76	4.43 ± 1.29
FSH (mIU/ml)	4.54 ± 1.30[a]	5.52 ± 3.30	4.45 ± 1.84	4.69 ± 1.62
LH (mIU/ml)	4.87 ± 1.94	4.02 ± 1.83	2.51 ± 2.75	3.27 ± 1.72
E2 (pg/ml)	37.15 ± 70.71[b]	34.67 ± 48.97	25.70 ± 1.73	34.67 ± 1.58
DHEA-S (nmol/l)	362.2 ± 131.6	361.6 ± 175.6	350.0 ± 171.9	433.3 ± 154.4
17-OHP (ng/ml)	1.25 ± 2.02[c]	1.14 ± 2.71[c]	1.24 ± 2.34[c]	3.55 ± 1.42
Total T (nmol/l)	104.7 ± 1.5[c]	109.6 ± 1.4[c]	91.2 ± 1.6	81.3 ± 1.5
Free T (nmol/l)	1.69 ± 2.04	1.45 ± 2.13	1.70 ± 2.34	1.99 ± 1.98
17-OHP >2 ng/ml (%)	27/105 (25.7)[c]	11/40 (27.5)[c]	6/25 (24)[c]	25/25 (100)
Acne (%)	33/105 (31.4)	13/40 (32.5)	8/25 (32.0)	12/25 (48.0)
Seborrhea (%)	42/105 (40.0)	12/40 (30.0)	9/25 (36.0)	8/25 (32.0)
Hair loss (%)	20/105 (19.0)	9/40 (22.5)	5/25 (20.0)	4/25 (16.0)
FGS	11.02 ± 2.71	11.09 ± 2.75	10.66 ± 1.75	10.06 ± 0.6

PCOM, polycystic ovary morphology; OA, oligoanovulation; HA, hyperandrogenism.
Values are means ± SD. BMI, body mass index; AMH, antimullerian hormone; FSH, follicle-stimulating hormone; LH, luteinizing hormone; E2, Estradiol; Total T, total testosterone; Free T, free testesteron; DHEA-S, dehydroepiandrosterone-sulfate; 17-OHP, 17-OH-progesterone.
FGS, Ferriman–Gallwey score.
Significant difference: $p < 0.05$ compared with groups.
[a] PCOM+/OA−/HA+.
[b] PCOM−/OA+/HA+
[c] LOCAH.

levels than those with PCOM−/OA+/HA+ but these differences were not statistically significant ($p > 0.05$).

There were no statistically significant differences among the four groups in terms of free T, LH and DHEA-S levels. The mean total T level in PCOM+/OA+/HA+ and PCOM+/OA−/HA+ groups were higher than the mean total T level measured in LOCAH group ($p < 0.05$). The LOCAH group had significantly higher 17-OHP levels than other groups ($p < 0.001$). The mean FSH and E2 levels of LOCAH group were similar with other three groups. 25.7% of patients with PCOM+/OA+/HA+, 27.5% of patients with PCOM+/OA−/HA+ and 24% of patients with PCOM−/OA+/HA+ had basal 17-OHP levels above 2 ng/ml. There were no statistically significant differences among the four groups in terms of percent of acne, seborrhea, hair loss and mean FGS levels. Twelve patients (48%) with LOCAH presented with polycystic ovaries on transvaginal ultrasound examination.

The ROC curves of the serum AMH and 17-OHP concentrations in discriminating PCOM+/OA+/HA+ from LOCAH were depicted in Fig. 2. The areas under the curves (AUC) were 0.806 (95% confidence interval [CI], 0.725–0.888), 0.911 (95% [CI] 0.857–0.966), respectively.

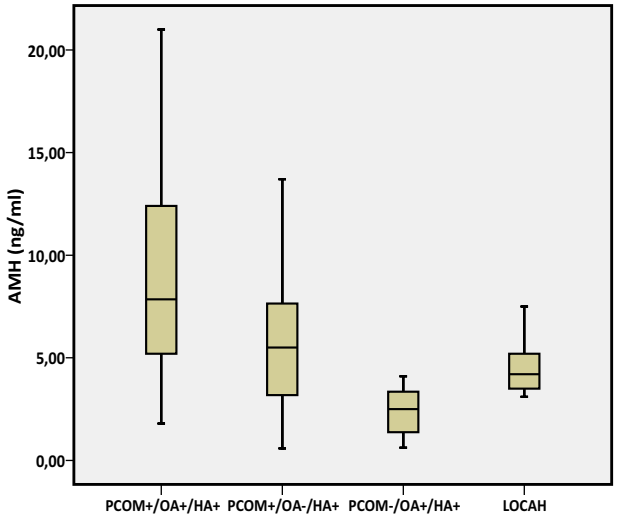

Fig. 1. The mean serum AMH levels of all groups.

Comments

Hyperandrogenic symptoms such as hirsutism, hair loss, seborrhea, acne vulgaris, weight gain, menstrual irregularities and infertility are well known manifestations of PCOS and LOCAH [6]. Thus, clinical presentations of LOCAH might be indistinguishable from the other clinical patterns of hyperandrogenemia, like PCOS.

The diagnosis of LOCAH can usually be made by measuring early morning serum 17-OHP levels. The elevation of basal 17-OHP is sometimes not effective and does not differ from that observed in patients with PCOS. Recently published study, Pall et al. showed that 25% of lean patients with PCOS, 21% of obese patients with PCOS, and 7% of controls had basal 17-OHP levels above 2 ng/ml [16]. Similarly, the present study showed that 25.7% of patients with PCOM+/OA+/HA+, 27.5% of patients with PCOM+/OA−/HA+ and 24% of patients with PCOM−/OA+/HA+ had basal 17-OHP levels above 2 ng/ml. Therefore, the aim of this study was to determine whether women with PCOS would be distinguishable from women with LOCAH on the basis of AMH levels. There were few studies that mentioned the difficulties in differentiating LOCAH patients from women with PCOS and the necessity of additional diagnostic measures for this situation. Pall et al. investigated whether differences in IR or hyperinsulinemia, or the presence of polycystic ovaries, may distinguish nonclassic adrenal hyperplasia from PCOS on behalf of this situation [16]. They documented that obese women with PCOS had significantly higher HOMA-IR and insulin levels than those with lean woman with PCOS, LOCAH patients and control subjects, but the incidence and degree of metabolic abnormalities was similar between lean women with PCOS and women with nonclassic adrenal hyperplasia.

Many studies have investigated the relationship of PCOS with serum AMH levels. Li et al. showed that the serum AMH levels in the hyperandrogenic pattern of PCOS patients were significantly higher than the non-hyperandrogenic patterns of PCOS [17].

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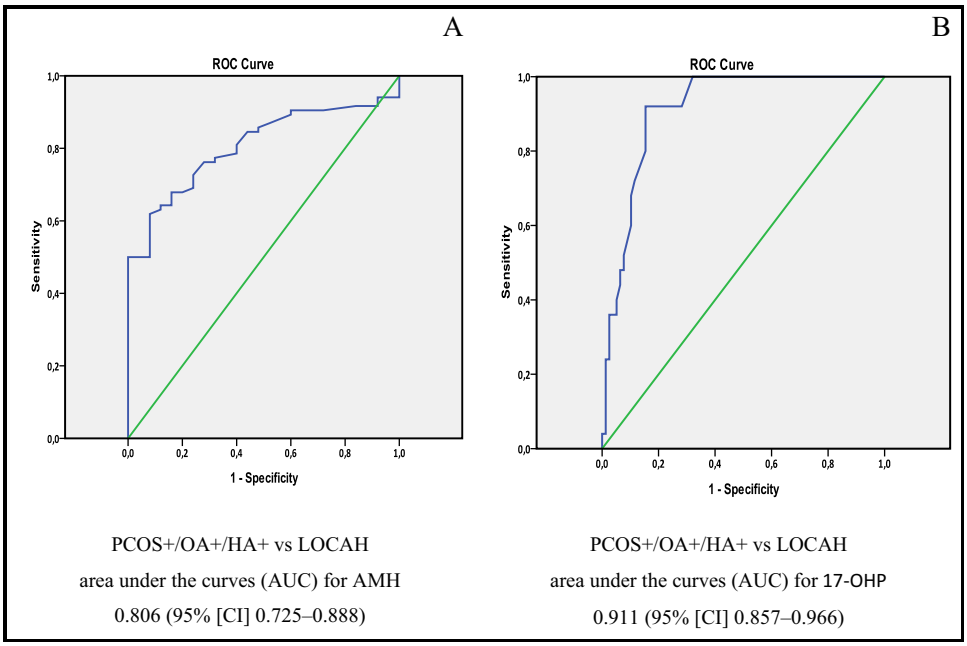

Fig. 2. Receiver operating characteristic (ROC) curves of serum AMH and 17-OHP in discrimination of PCOS+/OA+/HA+ vs LOCAH.

Likewise, Eldar-Geva et al. indicated that hyperandrogenism was associated with an extra increase in AMH [18]. Furthermore, according to Androgen Excess Society (AES), HA is considered to be a main indicator in the diagnosis of PCOS [19]. However, the present study showed that the highest AMH level was found in PCOM+/OA+/HA+ group and the lowest level of AMH was found in PCOM−/OA+/HA+ group. We assume that AMH levels are highly correlated with PCOM criterion.

We hypothesized that AMH levels of the LOCAH patients may be different from those of the PCOS patients because AMH is produced by ovaries. Although the mean AMH level in PCOM+/OA+/HA+ group was approximately two fold higher than the mean AMH level measured in LOCAH group, we did not find any differences between LOCAH and other phenotypes of PCOS on behalf of AMH levels.

AMH is not suitable for distinguishing LOCAH from all types of hyperandrogenic patterns of PCOS, but is only applicable for a specific subtype, such as PCOS patients with three main diagnostic criteria.

Limitations of this study include the small number of LOCAH patients and not evaluating the genotypic abnormalities observed in LOCAH. However, Witchel [3] suggested that genetic testing should not be considered a first-line diagnostic study in individuals suspected of LOCAH. Current screening panels assay for the 10–12 most common mutations, and may not be able to detect all mutations. Another limitation in this study was that the mean age of the PCOM+/OA+/HA+ and PCOM+/OA−/HA+ groups were higher than LOCAH group. However, Lie Fong et al. suggest that AMH is applicable only as a marker of the decline in ovarian reserve in women from 25 yr onward [20].

In conclusion, our study showed that AMH is not suitable for distinguishing LOCAH from all types of PCOS. AMH is only suitable for differentiating LOCAH from PCOS patients with three main diagnostic criteria. Therefore, ACTH stimulation test remains an essential clinical tool to diagnose LOCAH.

Conflict of interest

All authors have nothing to disclose.

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