Original Article

Serum Anti-Mullerian Hormone, New Marker in Diagnosis of Polycystic Ovary Syndrome among Women of Reproductive Age

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Abstract

Background: *Polycystic ovary syndrome (PCOS) affects up to one-fifth of women of reproductive age and causes anovulatory subfertility. Serum AMH (Anti-Mullerian Hormone) levels are stable throughout menstrual cycle, and are unmodified by pregnancy, gonadotropin releasing hormone treatment and administration of short-term oral contraceptives. These make AMH an ideal marker of ovarian reserve.*

Objective: *To analyze serum AMH levels among PCOS women of reproductive age to use AMH as a biomarker predictor along with other Rotterdam criteria.*

Materials and Methods: *In this cross-sectional study, a total of 200 women visiting in Gynae word at Combined Military Hospital, Dhaka were screened. Data were obtained from 100 normal healthy control and 100 PCOS newly diagnosed women aged 28.24 years (SD±4.84 years) meeting at least two of the Rotterdam criteria and specific inclusion criteria. Baseline variables, menstrual cycle length, hirsutism, sex hormones, TSH and serum AMH levels were analyzed during the follicular phase (1–5 days) of the menstrual cycle. Serum AMH was measured by electro chemiluminometric assay in Cobas 6000.*

Results*: A high serum AMH level (7.23±4.67 ng/ml) was recorded with normal sex hormone levels in PCOS patients. Women with PCOS had a significant mean deference for luteinizing hormone (p=0.04) and AMH levels (p=0.03) when compared with women of normal healthy control. Among PCOS patients BMI, oligomenorrhoea and infertility rate are more common than healthy controls.*

Conclusion: *An elevated serum AMH level can be used as a strong predictor to reflect the certainty of PCOS diagnosis among women of reproductive age when study concurrently with the other Rotterdam criteria.*

Key words: *Anti-mullerian Hormone, Marker, Polycystic Ovary Syndrome, Reproductive Age*

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Introduction

Polycystic *Ovary* syndrome (PCOS) is one of the most prevalent endocrine abnormalities for women of reproductive age. The prevalence of PCOS ranged from 6.11% (among the subjects visiting the gynecology outpatient department) to 92.16% (in subjects consulted for hirsutism). The prevalence was 29.9%–46.15% among infertile women.[1,2] A single study conducted among medical students reported a 37% prevalence.[3]

PCOS was initially described by Stein and Leventhal in 1935, although it had a history of at least a century beforehand. Stein and Leventhal initially defined this disorder as enlarged ovaries, hirsutism, obesity and anovulation. Rotterdam criteria described PCOS as a disorder with at least two of the clinical features: (1) oligo- or anovulation, (2) clinical or biochemical signs of hyperandrogenism and (3) polycystic ovaries with the exclusion of other etiologies (congenital adrenal hyperplasia, androgen, secreting tumors, Cushing's syndrome).1 Analysis of various hormones like FSH, LH, Prolactin, TSH to find out the pathology of anovulation is difficult. Due to pulsatile secretion of sex hormone their levels vary time to time. So sampling is difficult and troublesome. The use of serum AMH levels to detect ovarian dysfunction as a biochemical marker for early PCOS identification may be a good alternative as it is not altered during cycles. This study hypothesized that a serum AMH level above 4 ng/mL can be used to identify PCOS women of reproductive age as an auxiliary investigation to aid Rotterdam criteria.[4,5]

Polycystic ovarian syndrome is a heterogeneous syndrome affecting 5–10% of the female population.[6] According to Rotterdam Criteria The syndrome is characterized by at least

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two of the following three key components: a) Oligo-/amenorrhea; b) Clinical or biochemical hyperandrogenism; and c) Polycystic ovary morphology. In addition, women suffering from PCOS will often present metabolic disturbances, including obesity, insulin resistance and the metabolic syndrome.[7] Women with PCOS are known to have an excessive amount of preantral and small antral follicles in the ovaries and to have increased plasma AMH concentrations.[8,9] Anti-Mullerian hormone cannot be detected in resting primordial follicles, but the expression rapidly increases once a follicle reaches the preantral and antral stages (Figure 1). The highest level of AMH expression is seen in granulosa cells in preantral and small antral follicles of no more than 4 mm diameter and the expression disappears as follicles develop to the larger antral and preovulatory stages.[10] Anti-Mullerian hormone concentrations appear to correlate well with the severity of the syndrome and resistance to treatment. Hence, it has been proposed that AMH may be used as a marker for the extent of the disease. Additionally, AMH measurements have been found to offer relatively high specificity and sensitivity (92% and 69%, respectively) as a diagnostic marker for PCOS.8,[11-13]

Anti-Mullerian hormone (AMH) known as Mullerian-inhibiting substance (MIS) is a homodimer glycoprotein in nature belonging superfamily of transforming growth factor-β. The "p" arm (small) of chromosome 19 contains the gene for AMH.[14] By considering its structure, AMH is also linked with other members of transforming growth β (TGF-β)-like inhibin and factor-bone morphogenetic proteins (BMP), etc. which are also regulators of ovarian folliculogenesis.[15] Besides that, they show wide range of functions due to their extensive expression as ligand, whereas the expression of AMH is limited to the primary sex organs only and thus probably exerting its action only on the reproductive organs.[16] Emerging evidence reveals that the serum level of AMH is 2-3-fold increase in women suffering from PCOS compared to a normo-ovulatory control woman.[17,18] Despite substantial investigations and research work, the role of AMH remains elusive in PCOS, i.e. whether AMH is a significant marker of PCOS or a component accountable for PCOS.[19]

The appearance of AMH usually is observed from the primary follicle stage during folliculogenesis as this phase is FSH-dependent, the peak level of AMH expression is also observed from pre-antral and small antral follicles developed through the folliculogenesis and then gradually decreases along with the size of the follicle. The absolute absence of expression will be seen in the follicles diameter with more than 8 mm.[20,21] The expression of ovarian AMH and mRNA of anti-Müllerian hormone receptor type II (AMHRII) were attenuated by the action of FSH and estrogen during the differentiation of antral follicles and which is thought to be a crucial event required for follicular selection as AMH hinders the follicular development PCOS ovaries account for a huge presence of follicles with the diameter of up to 7 mm signifying the limited follicular growth at the moment when synthesis of AMH is highest.[22-25] AMH inhibits premature recruitment of follicles and follicular maturation. AMH will be suppressed if the follicles become large antral follicles followed by increased FSH (follicle-stimulating hormone) sensitivity leading to greater production of estrogen followed by two other physiological processes, i.e. selection of follicle and successive release of ovum as seen in the normal ovary. Thus, the inhibitory role of AMH during folliculogenesis may contribute to anovulation in PCOS. The reason for the raised AMH in women with PCOS is still largely unknown, but both excessive androgen production and insulin insensitivity may play a role.[26,27] Understanding the reason for the raised AMH might give clues concerning the mechanism of anovulation in PCOS.[28]

Materials and Methods

This was a cross-sectional study. The study was conducted in Gynae unit, Combined Military Hospital, Dhaka and Armed Forces Institute of Pathology (AFIP), Dhaka Cantonment. The study period was six months from 1st July 2024 to 30th December 2024. A total of 100 newly diagnosed PCOS patients and 100 normal healthy persons were recruited in this study. Variables were 1.AMH, 2.FSH, 3.LH, 4.TSH, 5.Prolactin. Inclusion criteria were Age between 18 and 45 years, PCOS diagnosed based on Revised Rotterdam Consensus 2003 criteria, Oligo-ovulation defined as length of menstrual cycle > 35 days or <10 periods per year, Clinical hyperandrogenemia is evidenced by hirsutism. Health controls met the following criteria: Regular menstrual cycle with an interval of 21-45 days, no medical history of hirsutism or severe acnel, no evidence of endocrine disease, no history of ovarian abnormalities and no history of ovarian or uterine surgery. Exclusion criteria were Primary amenorrhea (age ≥16 years), Hyperandrogenemia and infertility due to other known causes (primary hypothyroid, CAH, hyperprolactinemia).

Procedure of collecting data - a) Total 200 participants under Gynae in CMH, Dhaka was asked for proper history related to PCOS and other diseases, b) Data were collected by face-to-face interview with the participants by using a semi structured questionnaire in Bangla.

All necessary and relevant information regarding patients was recorded methodically and meticulously as far as possible.
A total of 200 patients were included in this study. Their blood samples were collected. Five ml of venous blood sample was taken in a plain tube from each patient. Laboratory assay: AMH and other hormones (follicle stimulating hormone, FSH; luteinizing hormone, LH;) by electro chemiluminometric assay in COBAS 601. Values of AMH were presented as nanograms per milliliters (conversion factor to pmol/l = ng/ml × 7.1). QC (quality control) was used in each assay run to assess precisions of the assay. Intraassay CV (co-efficient of variance) was 3.4 to 5.4% and interassay CV 4.0 to 5.6% for AMH assay. Ethical measures - Participation was voluntary,written consent was obtained after a brief of the study in Bangla to all participants,it was made clear to them that they were free to take part or refuse any part of the study,all the answers were kept confidential,every attempt was taken to conduct the interview privately.

After completion, data were checked, verified, edited, and coded. Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS) program, version 25. Values of AMH, FSH and LH samples in normal and PCOS patients were expressed as mean ± standard deviation. Student's

t-test for continuous variables were used. The reproductive characteristics of the PCOS women were compared using the independent T test between the groups with normal (≤4 ng/ml) and high AMH (>4 ng/ml) levels. A P-value of < 0.05 was considered as statistically significant.

Results

The results of this study are presented in two major sections. The first section includes the subject characteristics (age, sex, BMI, menstrual history etc.) and the second section deals with the results of the measured AMH, FSH, LH, TSH and Prolactin. Two hundred adult females of 18-45 yrs were recruited in our study. 100 were healthy control and 100 were PCOS patients. The mean age of healthy females 22.34 yrs and PCOS females 28.43 yrs. All participants were of Bangladeshi nationality.

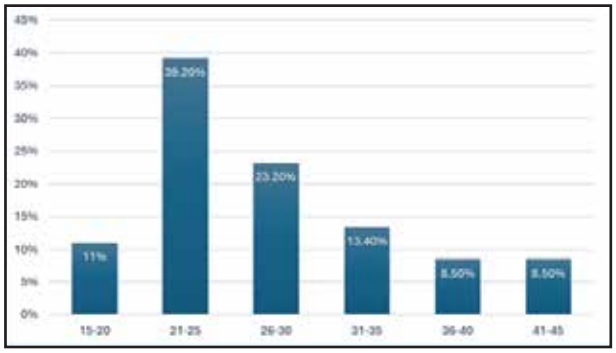

Figure 1: Age distribution in PCOS patients.

Baseline characteristics of subjects (as seen in Table-II) revealed statistically higher BMI (25.96± 11.99 vs. 23.02 ±7.1, kg/m2; p <0.05) in PCOS than that of control. Menstrual irregularity (86.3% vs. 13.7%; p<0.05) and infertility rate (68% Vs 32%,) were also markedly higher in the PCOS patients. As shown in Table-3.2, 87.8% PCOS subjects have hirsutism in different degrees but are absent in healthy controls.

Table I: Characteristics of the studied PCOS patients and control

Variables	PCOS n = 100	Controls n = 100
Age (mean ±SD,) years	28.43(8.73)	22.34(6.72)
Weight(kg)	86.07(10.15)	74.35(9.1)
Height(cm)	156.06(3.62)	153.2(5.8)
BMI (mean ±SD, kg/m ^{2})	25.96(11.99)	23.02(7.1)
Hirsutism	87.8%	
Menstrual disturbance	86.3%	41.7%
***Infertility**	68%	28.2%

*Analyzed over the married ladies only. Within parenthesis are percentages over column total (64control subjects and 72 PCOS patients were married)

Among the 100 women diagnosed with Polycystic Ovary Syndrome (PCOS), various physical examination parameters were recorded:

Body Mass Index (BMI): A significant proportion of the subjects were either overweight or obese. Specifically, 48% of the participants had a BMI in the overweight range (25–29.9 kg/m²), while 20% were classified as obese (BMI >30 kg/m²). Only 32% of the participants had a normal BMI (18–24.9 kg/m²).

Hirsutism: Evaluation of hirsutism, based on clinical scoring, revealed varying degrees of severity. Mild hirsutism (score 8–15) was observed in 43% of subjects, followed by moderate hirsutism (score 16–25) in 25%, and severe hirsutism (score 20–34) in 17%. Only 15% of the participants showed no signs of hirsutism (score <7).

Menstrual Cycle Patterns: Menstrual irregularities were prevalent among the participants. Oligomenorrhoea, defined as menstrual cycles lasting between 35 to 90 days, was the most common pattern and affected 70% of the women. Amenorrhea, or absence of menstruation for more than 90 days, was reported in 5% of the subjects. Only 25% of participants reported normal menstrual cycles (21–35 days).

Fertility Status: Infertility was a common concern among the PCOS subjects, with 68% identified as infertile. In contrast, 32% were considered fertile at the time of the study.

Table II: Reproductive hormonal Characteristics of the studied PCOS patients and control

Variable	PCOS **n=100 mean ±SD,**	Control **n=100 mean ±SD**
Follicle stimulating hormone	8.5 ± 10.7 mIU/ml	9.6 ± 3.17 mIU/ml
Luteinizing hormone	10.19 ± 5.20 mIU/ml	8.9 ± 7.36 mIU/ml
Prolactin	16.5 ± 7.6 ng/ml	13.2 ± 9.8 ng/ml
Anti Mullerian hormone	8.67 ± 1.25 mIU/ml	4.60 ± 0.32 mIU/ml
Thyroid Stimulating hormone	3.5 ± 1.92 mIU/ml	3.2 ± 2.3 mIU/ml

As depicted in Table II, FSH (<10.2mIU/ml), LH (<12.5mIU/ml), TSH (<4.20micro IU/ml), Prolactin (<29 ng/ml) were found to be normal among majority of the participants. AMH level was significantly higher (8.67 ± 1.25 vs. 4.60 ± 0.32, ng/ml; p<0.001) in the PCOS patients than that of controls.

However, when compared to age-group, this was significantly different between PCOS and controls in the age group 18-30 years (9.91 ± 0.71 vs. 4.52 ± 0.54, ng/ml; p<0.05) and age-group 31-45 years (8.28 ± 1.51 vs. 4.22 ± 0.68, ng/ml; p<0.05) shows in Table III.

Table III: Basal serum AMH levels in control women and in women with PCOS.

Group of subjects	Controls (n=100) AMH(ng/ml) ±(SD)	PCOS (n = 100) AMH (ng/ml) ±(SD)	p - value
Whole group	4.40 ± 0.41	9.21 ± 0.50	<0.005
Age group(years) n (control, PCOS)			
18 – 30 (49,60)	4.52 ± 0.54	9.91 ± 0.71	<0.005
31–45 (51,40)	4.22 ± 0.68	8.28 ± 1.51	<0.005

Analysis of reproductive hormone level among PCOS patients found that AMH is higher (>4 ng/ml) in 65% patients where other hormones are within normal level shown in Table IV.

Table IV: Reproductive Hormone level among PCOS women.

Variables	Groups	n (%)	Mean (SD)
Follicle stimulating hormone	Normal<10.2 mIU/ml	88	8.5 ± 10.7
	High>10.2 mIU/ml	12	
Luteinizing hormone	Normal<12.5 mIU/r	78	10.19 ± 5.20
	High>12.5 mIU/ml	22	
Prolactin	Normal<29 ng/ml	80	16.5 ± 7.6
	High>29 ng/ml	20	
Anti Mullerian hormone	Normal<4.0 ng/ml	35	8.67 ± 1.25
	High>4.0 ng/ml	65	
Thyroid Stimulating hormone	Normal<4.20 nmIU/L	90	3.2 ± 2.3
	High>4.20 nmIU/L	10	

Table V shows the comparison of reproductive characteristics between normal and high AMH levels among PCOS patients. The Independent t test showed a significant mean difference (p <0.05) for AMH and LH (p <0.05) reproductive hormone. Mean group Comparison between PCOS women with normal and high AMH levels showed a significant relationship (p <0.05) with LH but not significant with other hormone like FSH, Prolactin and TSH.

Table V: Comparison of reproductive characteristics between normal and high AMH level among PCOS patients.

Reproductive Outcome measure	AMH among PCOS women Mean ±SD,		Mean Difference (95% Cl)
	Normal<4ng/ml	High >4ng/ml	
Age	28.36 ± 9.60	29.45 ± 8.5	0.38 (-1.83, 5.31)
Average Menstrual cycle days	30.26 ± 15.51	42.21 ± 26.33	0.25 (-29.52, 7.76)
Follicle stimulating hormone	9.61 ± 3.96	11.02± 4.30	0.71 (-1.10, 1.56)
Luteinizing hormone	5.63 ± 1.96	13.18 ± 5.78	0.04* (-7.52, 0.35)
Prolactin	15.3 ± 11.05	16.98 ± 14.31	0.73 (-11.88, 8.54)
Thyroid Stimulating hormone	2.26 ± 0.82	1.87 ± 0.80	0.20 (-0.21, 0.98)

a; Independent t test, *p value.

Table VI shows subgroups of PCOS, and control subjects divided based on cut-off value of AMH at 4.0ng/ml. AMH ≥ 4.0ng/ml was considered positive in the diagnosis of PCOS. Thus 78 out of 100 out of 100 in the PCOS patients and 43 out of 100 controls could be labeled as positive. The calculated sensitivity was found to be 64.50% and specificity 78.33%

Table VI: Sensitivity and specificity of AMH for the diagnosis of PCOS holding cut-off as 4.00 ng/ml.

Group(s)	Anti -mullerian hormone (ng/ml)		Total
	<4.0ng/ml	**>4.0ng/ml**	
PCOS	78	22	100
Control	43	57	100
Total	121	79	100

PCOS: polycystic ovarian syndrome
Sensitivity = true positive / (all positive) × 100
= 78/ (78+43) × 100
= 64.50%
Specificity = true negative / (all negative) × 100
= 57/ (57+22) × 100
= 78.33%

Discussion

Polycystic ovary syndrome (PCOS), also known as hyperandrogenic anovulation (HA) or Stein–Leventhal syndrome. This chronic and heterogeneous disorder manifests itself as menstrual dysfunction, infertility, hirsutism, acne, and obesity.[23] PCOS is a heterogeneous disease and can manifest as a range of multifaceted problems, including various reproductive, cosmetic, cardiometabolic, and psychiatric conditions.[18] The prevalence of PCOS varies among ethnic populations and clinical presentation and different diagnostic criteria. It is estimated that British women aged 20–25 years have a prevalence of 33%, Finnish women aged less than 36 years have a prevalence of 21.6%, and the prevalence is 21% in New Zealandsssss and 23% in Australia among reproductive-aged women.[19-22] The prevalence of PCOS in South Asian women, especially in Pakistani women, is much higher (52%) than that in the white population (20–25% in the UK).[1]

PCOS has been known to be multifactorial. Anovulation and/or oligo-ovulation are the main underlying causes for infertility. Altered LH: FSH ratio, hyperandrogenemia, and hyperinsulinemia as well as insulin resistance – all had been thought to be linked to the probable cause of anovulatory cycles. But in the past decade much attention has been concentrated on AMH in the context of PCOS. AMH is synthesized as a pro-hormone. After its secretion, it activates. Ovarian granulosa cells are the only source of AMH, their concentration is inversely proportional with age, and it becomes faint after the menopause.[26-28] Anti-Mullerian hormones are secreted by the growing follicles; primary, secondary, preantral and small antral follicles. The AMH secretion increases as the follicles develop (shown in the figure by thickening of the arrows). The highest level of AMH secretion is by pre-antral and small antral follicles. Anti-Mullerian hormone has two major mechanisms of action in the ovary, firstly, AMH inhibits the initial recruitment of primary follicles from the resting pool of primordial follicles (step 1); and secondly, AMH inhibits the sensitivity of antral follicles to follicle-stimulating hormone (FSH) during cyclic recruitment (step 2). Anti-Mullerian hormone thereby prevents premature depletion of the follicle pool.

The present study was conducted to evaluate serum Anti-Mullerian Hormone (AMH) levels and their impact on PCOS patients compared to control subjects among women of reproductive age. Our findings demonstrated that AMH levels were significantly higher in PCOS patients than in controls, which aligns with previous studies. For instance, Pigny et al. and Laven et al. also reported elevated AMH levels in women with PCOS due to an increased number of small antral follicles.[29,30] This study supports the observation that PCOS women often have an increased menstrual cycle length with elevated serum AMH levels, potentially placing them at greater risk for menstrual disturbances. Serum AMH, being highly correlated with antral follicle count, has been considered a reliable surrogate marker for ovarian reserve and dysfunction. Dewailly et al. emphasized the role of AMH in identifying oligo/an-ovulation in PCOS and proposed that AMH could be an effective marker for diagnosing PCOS, especially in adolescents where the clinical symptoms may be ambiguous.[31] Our results also indicated that PCOS patients were more overweight (48%) and had higher rates of infertility (68%) compared to the control group, a trend that has also been documented in studies by Carmina and Lobo, and Pasquali et al., linking obesity and metabolic syndrome with PCOS.[32,33] The sensitivity and specificity of serum AMH for detecting PCOS in patients aged 18–45 years in our study were 67% and 78.33%, respectively, using a cut-off value of 4 ng/ml. This cut-off aligns with findings by Iliodromiti et al., who suggested that an AMH threshold between 3.5 and 5 ng/ml can be useful for diagnosing PCOS, although optimal values may vary slightly by population and assay method.[34] Higher AMH levels in PCOS patients are believed to result from a larger pool of small antral follicles, as granulosa cells of these follicles produce AMH. The inhibitory role of AMH on follicle recruitment and aromatase activity may lead to follicular arrest, contributing to anovulation. This mechanism has also been discussed by La Marca and Volpe, supporting the hypothesis that excess AMH disrupts follicular maturation.[35]

We observed a statistically significant correlation between serum AMH and LH ($p = 0.04$), and a trend toward higher LH secretion with increased AMH levels ($p = 0.03$). This is consistent with findings by Catteau-Jonard et al., who proposed that AMH may enhance LH receptor expression in granulosa cells, potentially contributing to the LH hyper-secretion commonly seen in PCOS.[36] Hyperandrogenism, a core feature in the Rotterdam criteria for PCOS diagnosis, was frequently seen in our cohort, especially in women with higher BMI. Clinical signs included hirsutism and acne, which were positively correlated with elevated AMH levels. These findings align with studies by Azziz et al., who observed a high prevalence of hyper androgenic symptoms in women with PCOS, particularly those with elevated AMH and BMI.[37] Our study strengthens the evidence that AMH can be a useful biochemical marker for the diagnosis of PCOS, especially when combined with clinical features as defined by the Rotterdam criteria. However, further multi center studies with larger cohorts are necessary to standardize AMH thresholds and validate its diagnostic utility across diverse populations.

Limitation of the study

- The phenotypic presentation of PCOS women can be altered by baseline variables such as age, BMI, physical activity level, and dietary patterns, which limits the finding of this study.
- The potential limitation of our data includes the small sample size and purposive sampling of women visiting the outdoor fertility center.
- This study did not compare the serum AMH levels with age and BMI matched non-PCOS women to estimate the normal ranges and trends.
- Considering the cross-sectional nature of the study, the participants were not observed over a longer period with follow-up biochemical analysis of serum AMH and other reproductive hormones to make an intra-rater comparison.

Conclusion

An elevated serum AMH level can be used as a strong predictor to reflect the certainty of PCOS diagnosis among women of reproductive age when study concurrently with the other Rotterdam criteria. The use of serum AMH is probably easier to perform than the use of an ultra-sonogram to estimate ovarian reserves. PCOS remains a syndrome, and as such, no single diagnostic criteria are sufficient for clinical diagnosis. This study used the Rotterdam criteria for PCOS identification and tried to use serum AMH levels as an auxiliary method for the identification of ovarian dysfunction.

This study further strengthened the established theory of using serum AMH as a marker in diagnosis of PCOS. Serum FSH, LH, Prolactin, and TSH are also done during PCOS diagnosis. This study shows Serum AMH is high in most PCOS patients and has a positive relation with raised LH which is statistically significant. So, Serum AMH can be used as a biochemical marker along with Rotterdam's criteria. The sensitivity and Specificity of Serum AMH are respectively 67% and 78.33%. This study, although is not representative of population of Bangladesh however, the findings reflected certain distinctiveness which will help to conduct further study on early detection of PCOS.

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