Investigation the Relationship between Spermatogenesis and the Levels of Some Hormones in a Sample of Infertile Iraqi Males with Azoospermia and Oligospermia

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Abstract

The objective of this study was to assess the levels of antiMullerian hormone (AMH), Follicle stimulating hormone (FSH), testosteron, luteinizing hormone (LH), and estradiol (E2) as markers of spermatogenesis between fertile and infertile males. This study was conducted at (AlRamadi Teaching Hospital for maternity and children) and included 136 males partners of infertile couples. Samples were classified according to the WHO criteria of semen analysis into three groups; Azoospermia, Oligospermia, and normal(control). Assay levels of these hormones were made in the serum and the semen of each sample. The results showed that the level of AMH in serum samples was non-significantly decreased ($P>0.05$) in both Azoospermia and Oligospermia groups compared to control (6.74 ± 3.21 ng/ml and 6.01 ± 0.25 ng/ml *vs*. 12.81 ± 7.69 ng/ml). While FSH and LH levels, there were non-significantly($P > 0.05$) increased in both azoospermia and Oligospermia compared to control (FSH: 12.11 ± 8.14 mIU/ml and 7.22 ± 6.06 mIU/ml*vs*.3.14 ± 1.27 mIU/ml; LH: 10.04 ± 7.23 mIU/ml and 7.62 ± 3.30 mIU/ml *vs*.5.57 ± 2.03mIU/ml).In addition, there were non- significant differences ($P> 0.05$) of testosterone hormone level among the studied groups. Such, there was a non-significant decreased level of E2 ($P> 0.05$) in both Azoospermia and Oligospermiagroups compared to control (170.49 ± 37.57 ng/ml and 137.20 ± 53.92 ng/ml *vs*. 194.91 ± 41.66 ng/ml).İn contrast to semen samples, the results of AMH showed a significant ($P< 0.05$) decreased level in both Azoospermia and Oligospermia compared to control (8.24 ± 4.17 ng/ml and 8.85 ± 4.79 ng/ml *vs*. 13.33 ± 0.77 ng/ml). FSH level was non-significant ($P> 0.05$) increased in Azoospermia and Oligospermia compared to control (3.97 ± 1.91 mIU/ml and 4.09 ± 2.50 mIU/ml *vs*. 2.47 ± 1.29 mIU/ml). Such, there were non-significant ($P> 0.05$) increased levels of LH and testosterone hormones in both Azoospermia and control compared to Oligospermia (9.35 ± 6.02 mIU/ml and 5.99 ± 4.43 mIU/ml *vs*. 4.06 ± 2.82 mIU/ml), and significant ($P< 0.05$) increased level of E2 in both Azoospermia and Oligospermia compared to control (241.36 ± 35.32 ng/ml and 220.86 ± 48.96 ng/ml *vs*. 170.71 ± 73.52 ng/ml).The study concluded that the AMH, FSH and E2 hormones levels in serum and semen samples may have been associated with the sperm production and may be a good marker for spermatogenesis, as well as Sertoli cell development. In addition, the age played a critical role in the level of the studied hormones.

Keywords: spermatogenesis, Azoospermia, Oligospermia, AMH, FSH, LH, Testosterone, E2, sex hormones.

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التحري عن العلاقة بين عملية تكوين النطف ومستويات بعض الهرمونات في عينة من الذكور العراقيين العقيمين عديمي وقليلي النطف

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الخلاصة

ان الفكرة من هذه الدراسة هو تقييم مستويات كل من هرمون مضاد مولر (AMH) ومحفزالجريبات (FSH) والملوتن (LH) و الشحمون (Testosterone) واستراديول (E2) كمؤشرات لعملية توليد الحيامن (spermatogenesis) في بعض الذكور العراقيين العقيمين وغير العقيمين. اجريت هذه الدراسة في مستشفى الرمادي التعليمي للنسائية والاطفال وشملت ١٣٦ ذكرا عقيما. تم تصنيف عينة الدراسة تبعا لمعايير منظمة الصحة العالمية لتحليل البلازما الى قليلي النطف Oligospermia و عديمي النطف Azoospermia والاشخاص الاصحاء Normospermia الذين يمثلون مجموعة السيطرة (Control). تم قياس مستويات تلك الهرمونات في المصل والسائل المنوي. وجدت الدراسة ان مستوى هرمون مضاد مولر (AMH) قد انخفض في مصل عديمي وقليلي النطف Azoospermia and Oligospermia بشكلغير معنوي (0.05 $P>$)مقارنة بمجموعة السيطرة ($vs.$ 6.01±0.25 ng/ml and 6.74±3.21 ng/ml) 12.81±7.69 ng/ml. بينما مستويات هرموني محفز الجريبات (FSH) والملوتن (LH) ازدادت بشكل غير معنوي (0.05 $P>$) في عديمي وقليلي النطف (FSH: 12.11±8.14 mIU/ml and 7.22±6.06 mIU/ml $vs.$ 3.14±1.27 mIU/ml; LH: 10.04±7.23 mIU/ml and mIU/ml $vs.$5.57±2.03mIU/ml 7.62±3.30).اضافة الى ذلك، لم تكن هنالك اختلافات معنوية (0.05 $P<$)في مستوى هرمون الشحمون بين المجاميع المدروسة. كذلك كان هناك انخفاضا معنويا (0.05 $P<$)في مستوى هرمون الاستراديول لدى عديمي وقليلي النطف مقارنة بالسيطرة (٣٧.٥٧±١٧٠.٤٩ ng/ml and 194.91±41.66 ng/ml $vs.$ 137.20±53.92 ng/ml). بالمقابل بالنسبة لعينات السائل المنوي، اوضحت النتائج ان هرمون مضاد مولر انخفض معنويا (0.05 $P<$)في عديمي وقليلي النطف مقارنة بالسيطرة (٤.١٧±٨.٢٤ ng/ml and 8.85±4.79 ng/ml $vs.$ 13.33±0.77 ng/ml). الهرمون المحفز للجريبات ازداد بشكل غير معنوي (0.05 $P>$) في عديمي وقليلي النطف مقارنة بالسيطرة (١.٩١±٣.٩٧ mIU/ml and 4.09±2.50 mIU/ml $vs.$ 2.47±1.29 mIU/ml). وكذلك كان هناك زيادة غير معنوية (0.05 $P>$)في مستويات هرموني الملوتن والشحمون في عديمي وقليلي النطف مقارنة بالسيطرة (٦.٠٢±٩.٣٥ mIU/ml and 5.99±4.43 mIU/ml $vs.$ 4.06±2.82 mIU/ml)، وزيادة معنوية (0.05 $P<$) في مستوى هرمون الاستراديول في عديمي وقليلي النطف مقارنة بالسيطرة (٣٥.٣٢±٢٤١.٣٦ ng/ml and 220.86±48.96 ng/ml $vs.$ 170.71±73.52 ng/ml). استنتجت الدراسة ان مستويات الهرمونات مضاد مولر ومحفز الجريبات والملوتن والشحمون والاستراديول في المصل والسائل المنوي لها ارتباطات بعملية انتاج الحيوانات المنوية وربما تكون علامة جيدة لعملية تكوين الحيوانات المنوية ونمو خلايا سيرتولي كما لعب العمر دورا حرجا في مستوى الهرمونات المدروسة.

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Introduction

Infertility as described by the WHO, is a reproductive system disease that characterized by the failure to achieve a clinical pregnancy after 12 months or more of regular unprotected sexual intercourse[1]. İt is a health problem affecting 32.2% of the general population. There is a growing interest in male infertility due to the decrease in semen quality in young healthy males worldwide [2]. Male infertility constitutes 25% of couples participating in fertility clinics and at least 50% of pairs requiring assisted reproductive techniques [3, 4].

Many factors such as Lifestyle, Diabetes, Obesity, Hormonal diseases, Testis Trauma, Cryptorchidism, Varicocele, Genitourinary Infections, Ejaculatory Disorders, Chemo/ Radiotherapy and Surgical Treatments are negatively affect on sperm quality [5, 6]. In addition, Chromosomal Abnormalities and Single Gene Mutations, which are effective in different stages of male physiological processes such as hormonal homeostasis, spermatogenesis and sperm quality, are estimated to constitute 10-15% of infertile cases. Forthermore, Idiopathic or Unexplained infertility accounts for only 60-75% of male infertile cases, giving little information about the underlying mechanism that regulates spermatogenesis and sperm function[7,8].

AMH formation is known for a long time that the male fetus Müllerian channels have been regressed[2].AMH is a member of the superfamily known as tissue growth factors and differentiation factors [3].Visser ve diğ demonstrated that the AMH expressed by testis during male gender differentiation continues to be secreted in adulthood [6].AMH is an indicator of Sertoli cells proliferation and protein synthesis activity in response to FSH before puberty, and is also a useful indicator of FSH activity in assessing testicular function in prepubertal men.Gonadotropins cause the production of protein hormones such as inhibin B and anti-Mullerian hormone (AMH) from Sertoli cells.Induction of Sertoli-germ cell interactions by inhibin B has been shown to play an important role in spermatogenesis [7].Spermatogenesis is initiated by the endocrine effect of gonadotropins [8].The AMH concentration in the seminal plasma shows that infertile oligozoospermic men have significantly lower levels of sperm concentration and mean testicular volume when compared to healthy volunteers [9].Another male reproductive hormone that plays a role in spermatogenesis is the testosterone that produced by Leydig cells under the effect of LH [10].Spermatozoan egg fertilization capability is largely due to the presence of compact chromatin which protects them during transport through the male and female reproductive tract [11], as well as the robust epididymal function that determines sperm maturation and motility [12].

Among patients with spermatogenic impairment, obstructive azoospermia and nonobstructive azoospermia patients are defined using FSH concentrations. However, elevated FSH levels (> 10 mIU / mL) do not always confirm the sperms are absent in the testes. Testicular biopsy is performed to confirm the presence or absence of sperm in such patients[10].

The aimof current study was to assess the anti-Mullerian hormone (AMH), Follicle stimulating hormone (FSH), testosteron, luteinizing hormone (LH), and estradiol (E2) as markers of spermatogenesis among fertile and infertile males as well as to examine the relationship among FSH, AMH, LH, testosterone and E2 in males.

Methodology

This work was planned as a sample control cross-sectional association study. The necessary approvals and permits for the study as well as the samples of the study were obtained from Al-Mulla Central Laboratory in AlRamadi teaching hospital for maternity and chidren.The study involved 136of voluntary male cases with age range 18-50 years who applied for infertility treatment. The cases were divided into three groups: fertile normozoospermia (n = 20), azoospermia (n = 24), Oligospermia (n = 24) (total n=68*2=136). According to age which were: 18-35 yrs. and 36-50yrs ,the medical history of the cases were taken from available hospital reports. Semen analysis was performed by professional lab technician and the results were reported according to WHO criteria [1].

Preparation and storage of specimens

Blood samples for biochemical analysis were taken at room temperature for 25 min. Samples of blood as well as semens were centrifuged for (5 min) at 1500 rpm.The serum and semen samples that obtained after centrifugation were labeled with eppendorf tubes and stored at -80 ° C until used.

Biochemical parameters examinations

The estimation of AMH concentration assessed with commercial ELISA kit (Ansh-US), testosterone levels with Biocore Diagnostik GmbH, Germany.While, FSH, LH and E2 were determined according to the protocol described by the manufacturer (Monobind inc, USA).

Statistical analysis

Statistical analysis were performed by using the IBM SPSS statistical software (SPSS for Windows ™, version 11.5 (SPSS Inc., Chicago, Illinois, USA). The data were expressed as mean and standard deviation (SD). Independent sample t-tests, ANOVA (one way variance) and the Tukey post-hoc test analysis were used to statistically evaluate differences between the studied groups.

Results

In the present study, the measurement results and statistical evaluations of Oligospermia, azoospermia and controls as well as the measurment of AMH, FSH, LH, Testosterone, E2 levels in blood serum and seminal plasma samples were given in Tables-(1 and 2), respectively.

The results of AMH serum level assessment in the age range 18 – 35 years showed that there were non-significant decreased level in azoospermia and Oligospermia compared to control.While there was a significant increase level in Oligospermia compared to Azoospermia. İn contrast, the results of 36 – 50 years group showed a significant increased level in Azoospermia compared Oligospermia and control, but there was no significant difference between Oligospermia and control. While, the total level of AMH showed a non-significant decreased level in both Azoospermia and Oligospermia compared to control Table-1.

The results of FSH hormone for the 18 – 35 years that demonstrated in Table-1 showed non-significant increased level in both Azoospermia and Oligospermia compared to control, also there was a non-significant increased level in Oligospermia compared to Azoospermia group. The same results appeared in 36 – 50 years group and the total patients group Table-1.

The results of LH hormone level in the sera of 18 – 35 years aged group showed a significant increased level in Azoospermia compared to control, and non-significant increased level in Oligospermia compared to control. In addition, there was no significant difference between Azoospermia and Oligospermia groups Table-1. While, the results showed a non-significant increased level in both Azoospermia and Oligospermia compared to control in both 36 – 50 years and total patients group. In addition, there was no significant difference between Azoospermia and Oligospermia groups Table-1.

The level of testosterone hormone in 18 – 35 years aged group showed a non-significant decreased level in Azoospermia group compared to control. The same results appeared in the total patients groups compared to control. In addition, there was a non-significant increased level in Oligospermia group compared to control, and there was no significant difference between the both patients groups Table-1.

While, the results of E2 hormone level in the sera of all the studied groups showed non-significant decreased level in Azoospermia and Oligospermia groups compared to control Table-1.

Table-2 showed the results of the studied hormones in semen samples. The level of AMH was non-significantly decreased in both Azoospermia and Oligospermia groups compared to control in 18 – 35 years aged group. While in 36 – 50 years aged group, there was a significant increased level in Azoospermia group compared to both controls and Oligospermiagroups. İn addition, there was a significant decreased level in Azoospermia compared to control, and non-significant difference between Oligospermia group and both Azoospermia and control groups in the total group.

Also, the results of FSH hormone showed a non-significant increased level in Oligospermia group compared to Azoospermia and control group, and a non-significant increased level in Azoospermia compared to control in all studied groups Table-2.

The results of LH hormone reffered to a significant increased level in Azoospermia group compared to both controls and Oligospermia groups of the 18 – 35 years aged group. While, there was a non-significant increased level in Azoospermia group compared to both controls and Oligospermia groups in the total group. Such, there were no significant differences between the studied groups for the 36 – 50 years aged group Table-2.

The results of testosterone hormone reffered that a non-significant decreased level in both Azoospermia and Oligospermia groups compared to controls for the 18 – 35 years aged group. While, there was a non-significant increased level in Azoospermia group compared to both controls and Oligospermia groups for both the 36 – 50 years aged group and the total groups Table-2.

Finally, the results of E2 hormone reffered that a non-significant increased level in both Azoospermia and Oligospermia groups compared to controls for the 18 – 35 years aged group. While, there was a significant increased level in Azoospermia group compared to both controls and Oligospermia groups for the 36 – 50 years aged group. Such there was a significant increased level in both Azoospermia and Oligospermia groups compared to controls for the total groups Table-2.

The results of our study reffered to correlation among hormones levels that studied in serum and semen. AMH levels in serum were significantly (p<0.01) corrlated with Testosteron levels in semen.

FSH levels in serum were significantly (p<0.01) correlatied with LH levels in semen. E2 levels in serum were significantly (p<0.01) correlated with FSH levels in semen Table-3.

Table 1- AMH, testosterone, FSH, LH, E2 levels in azoospermia, Oligospermia and control groups distributed according to age in serum samples.

Variable	Age group (years)	Control (N=20)	Azoospermia (N=24)	Oligospermia (N=24)
AMH (ng/ml)	18-35	**13.84±7.21[a]**	**4.63±1.39[ab]**	**6.48±2.03[b]**
	36-50	**1.68±0.25[b]**	**9.09±3.03[a]**	**2.46±0.96[b]**
	Total (18-50)	12.81±7.69[a]	6.74±3.21[a]	6.01±0.25[a]
FSH (mIU/ml)	18-35	**3.0±1.13[b]**	**12.28±8.60[a]**	**7.21±6.27[ab]**
	36-50	4.69±1.77[a]	11.94±7.72[a]	7.32±4.37[a]
	Total (18-50)	**3.14±1.27[b]**	**12.11±8.14[a]**	**7.22±6.06[ab]**
LH (mIU/ml)	18-35	**5.50±1.93[b]**	**8.15±2.85[a]**	**7.68±3.49[ab]**
	36-50	6.22±3.11[a]	12.16±9.72[a]	7.17±1.07[a]
	Total (18-50)	5.57±2.03[a]	10.04±7.23[a]	7.62±3.30[a]
Testosteron (nmol/l)	18-35	234.51±130.18[a]	191.69±81.86[a]	373.09±192.54[a]
	36-50	243.15±70.03[a]	207.17±23.64[a]	201.34±44.47[a]
	Total (18-50)	235.24±125.93[a]	199.01±61.76[a]	353.24±281.89[a]
E2 (ng/ml)	18-35	194.26±42.83[a]	171.25±49.63[a]	130.57±51.53[a]
	36-50	**201.97±27.13[a]**	**169.64±16.47[b]**	**189.87±43.07[b]**
	Total (18-50)	**194.91±41.66[a]**	**170.49±37.57[a]**	**137.20±53.92[b]**

Different letters reffered to a significant differences (*P*< 0.05).

Similar letters reffered to a non-significant differences (*P*> 0.05).

Table 2- AMH, Testosteron, FSH, LH, E2 levels in azoospermia, Oligospermia and control groups distributed according to age of semen samples.

Variable	Age group (years)	Control (N=20)	Azoospermia (N=24)	Oligospermia (N=24)
AMH (ng/ml)	18-35)	14.13±6.43[a]	5.15±3.85[a]	9.80±4.55[a]
	36-50	**1.37±0.16[b]**	**10.09±3.16[a]**	**3.85±2.31[b]**
	Total (18-50)	**13.33±0.77[a]**	**8.24±4.17[b]**	**8.85±4.79[ab]**
FSH (mIU/ml)	18-35	2.37±1.22[a]	3.92±1.90[a]	4.04±2.66[a]
	36-50	3.85±0.77[a]	3.99±1.96[a]	4.37±1.37[a]
	Total (18-50)	2.47±1.29[a]	3.97±1.91[a]	4.09±2.50[a]
LH (mIU/ml)	18-35	**5.83±4.44[ab]**	**11.18±7.41[a]**	**3.35±2.13[b]**
	36-50	8.33±3.92[a]	8.25±4.81[a]	7.80±3.17[a]
	Total (18-50)	5.99±4.43[a]	9.35±6.02[a]	4.06±2.82[a]
Testosteron (nmol/l)	18-35	272.47±112.06[a]	212.28±86.0[a]	239.54±37.25[a]
	36-50	305.39±76.63[a]	636.29±734.70[a]	203.33±52.40[a]
	Total (18-50)	274.53±110.11 [a]	477.34±615.61[a]	233.77±41.69[a]
E2 (ng/ml)	18-35	167.88±74.70[a]	229.71±37.42[a]	226.61±48.07[a]
	36-50	**203.71±45.00[b]**	**248.35±32.62[a]**	**190.51±44.27[b]**
	Total (18-50)	**170.71±73.52 [b]**	**241.36±35.32[a]**	**220.86±48.96[ab]**

Different letters reffered to a significant differences (*P*< 0.05).

Similar letters reffered to a non-significant differences (*P*> 0.05).

Table 3-Correlation coefficients between blood and seminal plasma AMH, testosterone, FSH, LH, E2 levels (Pearson correlation).

Variables		Seminal plazma				
		AMH	**FSH**	**LH**	**Testosteron**	**E2**
Blood plasma	**AMH**	0,358[**]	-0,176	-0,124	**0,343[**]**	0,042
	FSH	-0,006	0,337[**]	**0,424[**]**	0,015	0,019
	LH	-0,013	0,142	0,200*	-0,120	0,052
	Testosteron	0,136	-0,042	-0,092	0,084	0,124
	E2	0,022	**0,253[**]**	0,114	.0,055	0,157

[*]p<0.05, [**]p<0.01. n=136

Discussion

In general, the standered laboratory values of hormones in infertile men significantly agree somtimes and differ othertimes with our study.

According to Awni Kamal 2014, AMH levels between age group of (18-35) and (36-50) years were found to be (10.0 ± 16.6 ng / ml in serum) and (1.4 ± 2.3, ng / ml in semen) respectively for azoospermis patiens. In azoospermia patients compared to normospermic men, there were significant difference in FSH, testosterone and AMH concentrations. These findings may due to the regulation of AMH hormon beyond birth is complex and basal levels of AMH are independent of gonadotropinregulation[13].   These results are consistent with our study.

Other hand, Tuttelmann, F., et al., 2009 , reported that the level of AMH in men rises rapidly after birth, reaching the highest level in late infancy and gradually declining until puberty,AMH levels in serum were found to be 4.9 (1.8-13.6) ng / ml [14]. Concerning hormones, AMH correlated negatively with FSH in men with oligozoospermia and more pronounced in men with maldescended testes , AMH also correlated with total sperm count in men. Abstractly, AMH levels in serum are not affected by impaired spermatogenesis but are significantly correlated with spermatogenic. So that, AMH evaluation does not improve current clinical diagnostics. İn addition, Sertoli cell number, function, and maturation AMH should be evaluated with maldescended testes [14].These results are consistent with our study.

Goulis, D. G., 2009, had focused on serum FSH and AMH to predict the status of spermatogenesis in male testes [15]. Sharpe, R. et al., 2003, had also emphasized that AMH in oligospermic and azoospermic men might be an important marker in assessing testicular function in prepubertal boys, in that both Sertoli cell proliferation and protein synthesis activity are indicative of pre-pubertal FSH response [16].

İn our work, we reported that the AMH is an important factor for the normal development of male genitalia. Rey, R, et al. 1999, mentioned that serum AMH determination is clinically useful in assessing gonadal function [17].

Matuszczak, E., 2013, stated that Basal and FSH-induced AMH levels might be a useful predictive marker of gonadotropic cervical spermatogenic response in young patients with hypogonadotropic hypogonadism [18] . This fiding is compatible with our study .

Al-Chalabi, S., et al. 2012, conducted a comparison of serum biomarkers in normospermic, oligospermic and azoospermic men and found significant differences in FSH, testosterone and AMH concentrations in azoospermic men compared with normospermic men. It was also shown that AMH was significantly lower in men with oligospermia and azospermia rather than in control group [19]. This is consistent with our study except in the case of lack of sperm in the second group (36 - 50). The researcher took a single group (ie, 12 years to 50 years) as the level of the hormone AMH is high at the beginning of the youth as shown in our study Table-1, and the second reason was the use of the target group for frequent treatments (sexual stimulants).

Rey, R., 2000, had reported that serum AMH was a useful marker for follow-up of premature puberty or hypogonadotropic hypogonadism and follow-up of patients with gonadal stromal tumors [20] .

In addition, studies on seminal plasma of non-obstructive azoospermia men reported that AMH could be used as a marker of the presence of testicular spermatozoa when intracytoplasmic sperm injection is performed [18] . These results are parallel to our work.

Matuszczak, E., , et al. (2013), recorded that AMH was secreted by immature Sertoli cells (SC) and was responsible for the regression of Mullerian ducts in the male fetus as part of the sexual differentiation process and AMHs were at their lowest levels in the frst days afer birth but increased afer the frst week and it rised rapidly in concentration in boys during the frst month, reaching a peak level at about 6 months of age, and then slowly declined during childhood, falling to low levels in puberty[18].

According to Awni Kamal, R., (2014), FSH levels at age groups 18-35 and 36-50 years serum were found to be 20.4± 13.6 mlU/ml, respectively [13]. These results are consistent with our study.

According to Tuttelmann, F., et al., [14] , the level of serum FSH was 5.6± 3.5 mlU/ml in men. Concerning hormones, AMH correlated negatively with FSH in men with oligozoospermia as its clear in maldescended testes. The correlation between AMH and FSH was not found in men with normal sperm concentration.These results are consistent with our study [14].

According to Saleh, B., [21] , the FSH concentration that found in Oligospermia was 5.81 mIU/M and the LH concentration in the same subjects was 4.52 mIU/M. The control concentrations at the same age groups were   4.80 mIU/M and 3.67 mIU/M respectively, which were significantly higher than fertile controls (21). Concentrations of FSH and LH in our study were 7.21 mIU/M   and 7.68 mIU/M respectively, which are consistent with previously mentioned studies.

Zalata, A., et al. (2008), reported that serum FSH levels were significantly increased in infertile men compared with the normozoospermic group, and that serum testosterone levels were significantly lower than in the normozoospermic group and there was no statistically significant relationship with serum LH [22]. Elevated FSH levels are generally a reliable indicator of germinal epithelial damage and are usually associated with Azoospermia or Oligospermia. In addition, low testosterone levels may be İndicator of hypothalamic or pituitary hypogonadism [22].

In our study, we found that FSH and LH levels in the control and patients group were higher in the same age group as compared to the control group and the AMH and testeostron levels were lower than the control. Levels of AMH and testosterone in the Oligospermia and Azoospermia groups were found to have statistically significance.This means that they may be associated with these diseases. Burgu et al 2016, mentioned in their report that AMH was not produced in Oligospermia patients which is very interesting and supports our findings [23].

According to Awni Kamal 2014, LH serum levels at age groups 18-35 and 36-50 were found to be 9.3± 7.0 mlU/ml, respectively [13]. These results are consistent with our study.

According to Tuttelmann, F., et al., 2009, the level of serum LH was 3.7± 3.2 mlU/ml in men, these results are consistent with our study [14].

Sulthan, C., 2005, in his study, low testosterone and elevated LH were shown to be present in 20-30% of men. The underlying cause may be basal Leydig cell dysfunction [24]. In another similar study, infertile men were shown to have 18% lower Testosterone, 26% lower calculated Testosterone index and 34% lower Testosterone / LH levels compared to fertile control subjects [25].

According to Awni Kamal, 2014, Testosterone hormone levels in serum and sperm plasma at age groups (18-35) and (36-50) were found to be 327.7± 183.7 mlU/ml, respectively [13]. These results are consistent with our study.

According to Tuttelmann, F., et al., 2009, the level of serum E2 hormone was 74 ± 79 ml U/ml in men, these results are consistent with our study [14].

According to Awni Kamal, 2014, AMH levels in semen plasma at age groups (18-35) and (36-50) were found to be 1.4 ± 2.3 ng / ml [13]. These results are consistent with our study.

Al-Naqeeb, Asmhan A., 2015, observed a significant decrease in the level of seminal AMH in males with normozoospermia as compared to the males complaining of oligozoospermia. Also, a significant increase was noticed in the level of seminal AMH in males suffering from oligozoospermia as compared to normospermia and azoospermia. He mention that the AMH levels were not indicative of spermatogenesis and could not differentiate between fertile and infertile males[26].

Andersen, J. M. 2017, found that the AMH levels in seminal plasma were positively associated with sperm countand sperm motility [27]. We also revealed great individual differences in seminal levels of AMHcompared to serum levels.

Appasamy et al. 2007, Studied the relationship between serum AMH levels and semen characteristics and found a positive correlation between serum AMH levels and both sperm concentration and semen volume in a group of men undergoing infertility evaluation has been shown[9]. Al-Qahtani et al., 2005, Were used the same group of our study also and reported that serumAMH levels were lower in male infertility than in normal menwith oligozoospermia compared to controls [28].

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