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Seminal angiotensin II as a predictive factor of spermatogenic activity in non-obstructive azoospermia

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Abstract

Background: There is an unmet need for a non-invasive marker to predict spermatogenic potential and the likelihood of finding mature sperm in cases of non-obstructive azoospermia (NOA). Accordingly, we assessed the level of seminal angiotensin II (Ang II), which is suggested to be linked to sperm motility and count, and its relation to spermatogenic activity. **Methods**: A prospective case-control study included three groups: Group I, consisting of 30 male patients with NOA; Group II, consisting of 30 male patients with obstructive azoospermia (OA); and Group III, consisting of 30 healthy fertile males as the control group. **Results**: Seminal Ang II levels were significantly lower in OA patients compared to NOA patients ($p < 0.001$) and controls ($p < 0.001$). Additionally, angiotensin II levels were significantly lower among NOA patients than controls ($p < 0.001$). In NOA patients, the lowest Ang II average was observed in those with Sertoli cell-only syndrome (SCO) and the highest in those with late maturation arrest ($p \leq 0.05$). **Conclusions**: Our findings suggested that seminal Ang II may serve as a predictive marker for spermatogenesis and the presence of mature sperm in microsurgical testicular sperm extraction (micro-TESE) in cases of non-obstructive azoospermia.

Keywords

Azoospermia; Non-obstructive; Angiotensin II; Spermatogenic activity

Angiotensina II seminal como factor predictivo de la actividad espermatogénica en azoospermia no obstructiva

Resumen

Antecedentes: Existe una necesidad insatisfecha de un marcador no invasivo para predecir el potencial espermatogénico y la probabilidad de encontrar espermatozoides maduros en casos de azoospermia no obstructiva (ANO). En consecuencia, evaluamos el nivel de angiotensina II seminal (Ang II), que se sugiere está vinculada con la motilidad y el conteo de espermatozoides, y su relación con la actividad espermatogénica. **Métodos**: Se realizó estudio prospectivo de casos y controles incluyó tres grupos: Grupo I, compuesto por 30 pacientes masculinos con ANO; Grupo II, compuesto por 30 pacientes masculinos con azoospermia obstructiva (AO); y Grupo III, compuesto por 30 hombres fértiles saludables como grupo control. **Resultados**: Los niveles de angiotensina II seminal fueron significativamente más bajos entre los pacientes con AO en comparación con los pacientes con ANO ($p < 0.001$) y controles ($p < 0.001$). Además, los niveles de angiotensina II fueron significativamente más bajos entre los pacientes con ANO que entre los controles ($p < 0.001$). En los pacientes con ANO, el promedio más bajo de Ang II se observó en aquellos con síndrome de células de Sertoli únicamente (SCO) y el más alto en aquellos con detención de maduración tardía ($p \leq 0.05$). **Conclusiones**: Nuestros hallazgos sugieren que la Ang II seminal podría servir como un marcador predictivo de la espermatogénesis y la presencia de espermatozoides maduros en la extracción de espermatozoides testiculares mediante microscirugía (micro-TESE) en casos de azoospermia no obstructiva.

Palabras Clave

Azoospermia; No obstructiva; Angiotensina II; Actividad espermatogénica

1. Introduction

The absence of spermatozoa in the ejaculate is known as azoospermia. Two types of azoospermia exist: obstructive (OA) and non-obstructive (NOA) [1]. The primary cause of OA is epididymal pathologies or congenital anomalies resulting from obstructions in the testicular and genital duct system [2]. In contrast, NOA is characterized by a failure of the testicles to produce sperm [3].

To diagnose azoospermia and differentiate between OA and NOA, semen analysis, medical history, clinical examination, and specific indirect endocrine tests may provide clues. Nevertheless, testicular biopsy is currently the only reliable diagnostic technique. However, it is an invasive surgical procedure and entails several complications, including tissue damage, bleeding and chronic pain [3].

To reduce the need for testicular biopsies, provide a more precise evaluation of histopathological patterns, and enable improved treatment goals, there is an unmet need for a non-invasive marker test that predicts spermatogenic potential and the possibility of finding mature sperm in cases of NOA [1].

In the male urogenital tract, glands secrete a biological fluid known as seminal plasma (SP). Thus, research into the usefulness of non-invasive SP biomarkers in predicting successful sperm retrieval (SSR) is ongoing [4].

The renin-angiotensin system (RAS) is believed to play a potential role in spermatogenesis. Angiotensin II, the most active peptide of the RAS, is suggested to participate in controlling many biological processes, including reproduction [5]. Exposure of human spermatozoa to angiotensin II increases the proportion of motile sperm and promotes specific sperm motility characteristics [6]. Additionally, it plays a role in the physiological function of the epididymis [7].

Accordingly, this work aimed to assess the level of seminal angiotensin II in NOA and OA and its relation to spermatogenic activity.

2. Materials and methods

The study, started in September 2021, was conducted on 90 male patients divided into three groups recruited from the Andrology Clinic of Alexandria Main University Hospital. Group I included 30 male patients with NOA, Group II included 30 male patients with obstructive azoospermia, and Group III included 30 healthy fertile males as the control group. Cases of azoospermia were confirmed with two semen samples where semen analysis showed no sperm detected, even after centrifugation.

We excluded patients with any medical illness, systemic disease or drug intake that could affect testicular function, those who were on angiotensin II receptor blockers, and those who received ACE (angiotensin-converting enzyme) inhibitors.

Detailed history, general examination, clinical genital examination and semen analysis were conducted. We included azoospermic patients according to World Health Organization (WHO) criteria (absence of sperm in at least two different ejaculates, including the centrifuged sediment). Hormonal assessment was performed, including serum follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin and free testosterone, using chemiluminescence immunoassay (CLIA). The kits for FSH (Cat No: E-CL-H0760) and LH (Cat No: E-CL-H0115) were provided by Elabscience Company, USA (Website: www.elabscience.com). The kit for free testosterone (Cat No: TSF31-L01) was provided by Eagle Biosciences Company, USA (Website: www.eaglebio.com).

Patients were subjected to seminal alpha-glucosidase tests and quantitative fructose assessments to rule out obstruction. A testicular biopsy was performed unilaterally on the larger testis, and when the testes volumes were equal, the procedure was always performed on the right side. Briefly, the tunica vaginalis was opened following a midline scrotal incision. The testis was widely opened in an equatorial plane, revealing the testicular tissue. The remaining steps of the operation were carried out under a 16–25× operating microscope. The tubules were dissected, and tissue samples were collected in separate containers filled with culture medium for immediate examination by an embryologist. The surgery was concluded once all visible parenchymal regions were examined under a microscope or when additional dissection was deemed likely to endanger the testicular blood supply. The micro-TESE procedure was considered successful if at least one motile spermatozoon was retrieved. An additional sample was collected for histological examination, which was fixed in Bouin's solution.

Semen samples were taken from all subjects after 3–5 days of abstinence to measure angiotensin II according to the manufacturer's instructions. Semen samples were centrifuged for 15 minutes at 14,000 rpm at 4 °C. The supernatant was collected and stored at −20 °C until the time of assay. The level of angiotensin II in seminal plasma was detected using a human solid-phase competitive enzyme-linked immunosorbent assay (ELISA) following the manufacturer's instructions [8]. The kit (Cat. No: EH2627) was provided by Fine Test Company, China (Website: www.fn-test.com). Samples and standards were run in duplicates, and the color change was detected spectrophotometrically at 450 nm. After plotting the standard curve, the sample optical density (OD) was used to determine the target concentration in the samples.

All collected data were analyzed using the IBM SPSS software package version 20.0 (IBM Corp, Armonk, NY, USA). Quantitative data were labeled using range (minimum (Min) to maximum (Max)), mean and standard deviation (SD). The significance of our results was mediated at a level of 5%. The chi-square test was used to compare categorical variables between the three groups and corrected with the Monte Carlo correction when >20% of the cells had an expected count of <5. The *F*-test Analysis of Variance (ANOVA) was used to compare normally distributed quantitative variables between the three groups. The *post-hoc* test (Tukey) and the Kruskal-Wallis test were utilized for abnormally distributed quantitative variables, with *post-hoc* (Dunn's multiple comparisons test) for pairwise comparisons.

3. Results

The mean age was comparable across the three studied groups, with no statistically significant age difference ($p = 0.259$). In all three groups, the majority of men were smokers. A genital

examination was conducted on all subjects. Among NOA patients, 86.7% had normal-sized testes, while 100% of OA patients and controls had normal-sized testes (Table 1).

Concerning laboratory tests, no statistically significant difference was observed between the three groups in terms of serum FSH (p = 0.142), serum LH (p = 0.130), serum-free testosterone (p = 0.414), and serum prolactin (p = 0.930) (Table 2).

Angiotensin II level was measured in all groups, and its lowest value was among the OA patients. Angiotensin II levels ranged from 7.3 to 93.4 pg/mL with a mean of 45 pg/mL among the OA patients, from 21.8 to 289.2 pg/mL with a mean of 131.47 pg/mL among the NOA patients, while it ranged from 153.7 to 1420.8 pg/mL with a mean of 431.51 pg/mL among controls ($p < 0.001$) (Table 3).

The distribution of the studied cases according to testicular pathology in each group is shown in Table 4. In the NOA group, 9 patients were diagnosed with early maturation arrest at primary spermatocyte (30%), 5 patients were diagnosed with Sertoli cell-only (SCO) only (16.7%) [9], 7 patients (23.3%) were diagnosed with late maturation arrest, while 9 patients (30%) were diagnosed with mixed pathology. In the OA group, the pathology showed that all patients had mature sperms.

The average Ang II level in different testicular pathologies was 44.8 pg/mL in patients with SCO [9], 100.09 pg/mL in patients with early maturation arrest at primary spermatocyte, 165.79 pg/mL in patients with mixed pattern, and 189.59 in patients with late maturation arrest (Table 5).

The mean Ang II level was significantly higher in NOA patients with positive TESE results than in NOA patients with negative TESE results (196.57 ± 44.72 pg/mL *vs.* 88.07 ± 32.60 pg/mL, respectively; $p < 0.001$) (Table 6).

TABLE 1. Comparison between NOA patients, OA patients, and controls based on age, smoking and genital examination.

Age (yr)	Non-obstructive (n = 30)		Obstructive (n = 30)		Control (n = 30)		F	p
Min.–Max.	23.0–48.0		23.0–50.0		25.0–41.0		1.384	0.259
Mean ± SD.	31.37 ± 6.55		34.87 ± 9.03		31.60 ± 4.93			
Smoker	No.	%	No.	%	No.	%	χ^2	MC_p
Non-smoker	6	20.0	14	46.7	12	40.0	4.007	0.153
Smoker	24	80.0	16	53.3	18	60.0		
Genital examination	No.	%	No.	%	No.	%	χ^2	MC_p
Normal size testis (15–25 mL)	26	86.7	30	100.0	30	100.0		
One testis is small in size (7 mL)	1	3.3	0	0.0	0	0.0	3.278	0.713
Both testes are small in size (7–10 mL)	3	10.0	0	0.0	0	0.0		

F: one-way ANOVA test; χ^2: chi-square test; MC: Monte Carlo; Min.: minimum; Max.: maximum; SD.: standard deviation.

TABLE 2. Comparison between NOA patients, OA patients, and controls based on hormonal assay.

Hormonal assay	Non-obstructive (n = 30)	Obstructive (n = 30)	Control (n = 30)	H	p
Serum FSH (mIU/mL)					
Min.–Max.	1.34–27.60	1.37–8.50	1.60–9.40	3.910	0.142
Mean ± SD.	6.94 ± 5.39	4.29 ± 2.01	4.64 ± 2.53		
Serum LH (mIU/mL)					
Min.–Max.	3.10–20.10	2.40–9.51	4.90–8.20	4.075	0.130
Mean ± SD.	8.10 ± 3.61	6.88 ± 1.87	6.69 ± 1.17		
Free testosterone (pg/mL)					
Min.–Max.	53.90–279.00	78.20–253.50	62.80–299.10	1.768	0.413
Mean ± SD.	133.63 ± 52.90	136.51 ± 41.51	147.43 ± 57.07		
Serum Prolactin (ng/mL)					
Min.–Max.	4.20–18.50	4.90–13.10	5.80–18.40	0.144	0.930
Mean ± SD.	8.62 ± 3.38	8.34 ± 2.49	9.61 ± 4.41		

H: Kruskal Wallis test; FSH: follicle-stimulating hormone; LH: luteinizing hormone; Min.: minimum; Max.: maximum; SD.: standard deviation.

TABLE 3. Comparison of NOA patients, OA patients, and controls based on angiotensin II level.

Angiotensin II level (pg/mL)	Non-obstructive (n = 30)	Obstructive (n = 30)	Control (n = 30)	*H*	*p*
Min.–Max.	21.80–289.20	7.30–93.40	153.70–1420.80	41.742*	<0.001*
Mean ± SD.	131.47 ± 65.61	45.00 ± 30.49	431.51 ± 296.07		
Sig. bet. grps.		$p1 = 0.001^*$, $p2 < 0.001^*$, $p3 < 0.001^*$			

*H: Kruskal Wallis test; p: p-value for comparing between the three studied groups; p1: p-value for comparing between non-obstructive and obstructive; p2: p-value for comparing between non-obstructive and control; p3: p-value for comparing between obstructive and control; *: statistically significant at $p \leq 0.05$.*
Min.: minimum; Max.: maximum; SD.: standard deviation. Sig. bet. grps.: significance between groups.

TABLE 4. Distribution of NOA and OA patients based on testicular pathology in each group.

Testicular pathology	No.	%
Non obstructive (n = 30)		
SCO only	5	16.7
Early maturation arrest at primary spermatocyte	9	30.0
Late maturation arrest	7	23.3
Mixed patterns	9	30.0
SCO + early maturation arrest at primary spermatocyte	3	10.0
SCO + tubular hyalinization	4	13.4
Early maturation arrest + hypospermatogenesis	1	3.3
Tubular sclerosis + tubular hyalinization + interstitial hyperplasia	1	3.3
Obstructive (n = 30)		
Mature sperms	30	100.0

SCO: Sertoli cell-only.

TABLE 5. Distribution of NOA and OA patients based on testicular pathology in the NOA group.

Testicular pathology	No. of pattern	Average (Angiotensin II level)	Median (IQR)	*H*	*p*
SCO only	5	44.80	53.30 (28.40–58.90)	21.669*	<0.001*
Early maturation arrest at primary spermatocyte only	9	100.09	103.1 (83.30–110.4)		
Mixed patterns	9	165.79	147.4 (124.2–219.3)		
Late maturation arrest	7	189.59	185.4 (154.5–209.5)		

H: Kruskal Wallis test; SCO: Sertoli cell-only; IQR: Interquartile Range.
**: Statistically significant at $p \leq 0.05$.*

TABLE 6. Relation between Ang II level and results of TESE in NOA (wet smear) (n = 30).

Angiotensin II level (pg/mL)	Results of TESE		U	*p*
	Negative (n = 18)	Positive (n = 12)		
Min.–Max.	21.80–131.60	136.80–289.20		
Mean ± SD.	88.07 ± 32.60	196.57 ± 44.72	0.0*	<0.001*
Median (IQR)	98.25 (61.60–112.40)	193.00 (154.50–225.90)		

U: Mann Whitney test; Min.: minimum; Max.: maximum; SD.: standard deviation; TESE: testicular sperm extraction; IQR: Interquartile Range.
**: Statistically significant at $p \leq 0.05$.*

4. Discussion

To the best of our knowledge, this study is the first to investigate angiotensin II in both NOA and OA. In the present work, angiotensin II levels were measured in all groups, with the lowest value observed among OA patients ($p < 0.001$). This result was expected, as seminal angiotensin II is secreted locally from the male genital tract and is higher than serum angiotensin II levels in the blood [5]. Thus, any obstruction in the genital tract subsequently reduces all seminal fluid components, including seminal angiotensin II. Additionally, we observed that angiotensin II levels were significantly lower among NOA patients compared to controls ($p < 0.001$), with the lowest average angiotensin II level in NOA patients with Sertoli cell-only (SCO) syndrome and the highest in NOA patients with late maturation arrest.

Our findings indicated that low seminal angiotensin II levels are related to azoospermia, whether OA or NOA. We concluded that angiotensin II levels are related to spermatogenic activity and could predict the presence of mature sperm in micro-TESE in cases of NOA, supporting the previously suggested regulatory role of angiotensin II in male reproduction and its relation to male fertility status [10].

In a recent meta-analysis, Zhang *et al.* [11] reported several predictors for successful micro-TESE after failed initial micro-TESE in NOA patients, including younger age, small bilateral testicular volume, lower levels of FSH and LH, and histopathological findings of hypospermatogenesis. In contrast, patients with SCO were more likely to experience repeated failure in micro-TESE.

Regarding testicular size, we observed that all OA patients, controls, and the majority of NOA patients (86.7%) had normal-sized testes, with no statistical difference between the studied groups ($p = 0.713$). This suggested that there is no relationship between testicular size and azoospermia. Our findings suggested that testicular size is not a reliable indicator for diagnosing azoospermia or distinguishing between OA and NOA. Smaller testicular volumes are generally expected to be more prevalent in NOA patients; however, this could be attributed to the small sample size, where only 4 test subjects out of 30 had small testes.

Contrary to our findings, Huang *et al.* [12] attempted to differentiate between NOA and OA by hormonal profile and testicular size. They observed that among 156 NOA patients and 51 OA patients, the mean size of the bilateral testes was significantly smaller in the NOA group than in the OA group ($p < 0.0001$). These findings are consistent with those of Schoor *et al.* [13], who reported that testicular size among a total of 153 azoospermic men was an essential clinical indicator for discriminating between OA and NOA patients. Similarly, Shamohammadi *et al.* [14] found that testicular size was significantly greater in OA patients compared to NOA patients ($p < 0.05$). The differences in sample sizes, underlying pathology, and ethnic and racial differences may explain the varying results between our study and these previous studies.

We observed that serum FSH and LH were higher in NOA patients, although not statistically significant ($p = 0.142$ and 0.130, respectively) it may be attributed to the small sample size which can be considered one of the limitations of this study. Additionally, no statistically significant difference between OA patients, NOA patients, and controls regarding serum-free testosterone ($p = 0.414$) and serum prolactin ($p = 0.930$) was detected. Our findings are consistent with those of Ali *et al.* [1], who investigated seminal plasma biomarkers and their role in discriminating between OA and NOA. They found that serum FSH, LH, free testosterone, and prolactin were comparable in OA patients, NOA patients, and controls ($p > 0.05$).

However, our findings differ from those of other studies. Schoor *et al.* [13] reported that in NOA patients, serum FSH, LH and free testosterone were significantly higher compared to OA patients ($p < 0.01$), while serum prolactin levels were nearly equal in both groups ($p = 0.95$). Shamohammadi *et al.* [14] found that serum testosterone levels in OA patients were significantly higher than in NOA patients, while serum FSH and LH levels in OA patients were significantly lower than in NOA patients ($p < 0.05$).

This discrepancy may be related to different underlying pathologies in NOA patients, such as maturation arrest or SCO syndrome. Maturation arrest is associated with normal serum FSH levels, while SCO histopathology is linked to high serum FSH levels, leading to differences in mean FSH estimation. For example, maturation arrest was diagnosed in most of our studied samples compared to SCO, which could explain the normal mean FSH levels observed.

The main strength of our work lies in being, to our knowledge, the first study to investigate seminal angiotensin II as a marker in azoospermic patients. Additionally, this study's prospective, case-control design strengthens its findings by minimizing potential bias. However, the small sample size is the primary limitation of this work. A larger sample size would allow for a more comprehensive assessment of seminal Ang II as a predictive marker and provide greater statistical power to validate our findings. Another limitation is the lack of longitudinal follow-up, which would help determine the reproducibility of seminal Ang II measurements over time and their reliability in predicting micro-TESE outcomes. Future studies with larger sample sizes and a longitudinal design are recommended to confirm our results and establish clinical applicability.

5. Conclusions

Our data suggested that seminal angiotensin II might predict spermatogenic potential and the presence of mature sperm or haploid cells in cases of non-obstructive azoospermia, where a low level may contribute to male infertility. This supports the potential regulatory role of angiotensin II in male reproduction and its relation to male fertility status.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this study are available on reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

AT and TH—planned the research study. AT and NA—performed the research; analyzed the data and wrote the manuscript. IMA and TH—delivered support on clinical examination. WNR—delivered support on laboratory investigations. The manuscript's editing revisions were made with input from all authors. All authors reviewed and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The Faculty of Medicine, Alexandria University's ethical committee approved the study IRB NO: 00012098 (Expires 6-10-2022); FWA NO: 00018699 (Expires 21-01-2026). All subjects signed informed consent after explaining the nature and purpose of our work.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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