

Original Research Article

# The relationship between anogenital distance and benign prostate hyperplasia-related lower urinary tract symptoms

Rahul Chethan V\*

Assistant Professor of Urology, Deccan College of Medical Sciences, Owaisi Hospital and Research Centre, India

\*Corresponding author email: [Vrahulchetan@gmail.com](mailto:Vrahulchetan@gmail.com)

	International Archives of Integrated Medicine, Vol. 8, Issue 4, April, 2021.	
	Available online at <a href="http://iaimjournal.com/">http://iaimjournal.com/</a>	
	ISSN: 2394-0026 (P)	ISSN: 2394-0034 (O)
	Received on: 03-03-2021	Accepted on: 19-03-2021
	Source of support: Nil	Conflict of interest: None declared.
<b>How to cite this article:</b> Rahul Chethan V. The relationship between anogenital distance and benign prostate hyperplasia-related lower urinary tract symptoms. IAIM, 2021; 8(4): 75-78.		

## Abstract

**Background:** Anogenital distance is often used as biomarker of intra uterine androgen levels. The androgen may influence the benign prostatic hypertrophy and lower urinary tract symptoms subsequently. This was mainly undertaken to study the relation between the anogenital distance and the lower urinary tract symptoms.

**Material and methods:** A comparative study was undertaken among 50 LUTS patients and 50 controls in Urology department of a tertiary care hospital. All the cases were studied for International Prostate Symptom Score (IPSS), Prostate volume, Maximum flow rate ( $Q_{max}$ ), Prostate specific antigen (PSA), testosterone levels, height, weight were recorded. Two types anogenital distance was obtained AGDAP (from the centre of the anus to the cephalad insertion of penis) and AGDAS (from the center of the anus to the posterior base of the scrotum) were measured.

**Results:** The maximum flow rate was significantly higher in controls. This study had shown that the AGD was higher in cases than controls. The PSA levels were significantly higher in LUTS patients and also testosterone levels. The BMI adjusted values of AGD (AS) also was significantly lower in LUTS group than the controls.

**Conclusion:** The anogenital distance can be a marker for benign prostatic hyperplasia and this knowledge can be used for prevention.

## Key words

Benign prostatic hyperplasia, Anogenital Distance, Prostatic Specific Antigen, Testosterone, Prevention.

## **Introduction**

Anogenital distance (AGD) is the distance between the anus and sexual organs is often a dimorphic feature in human beings [1]. It is often used as a marker of genital development in animal model and humans, being longer in males [1, 2]. A number of studies had shown that AGD at birth reflects the androgen levels during the in utero development and predicts AGD during adulthood. Higher in utero androgen exposure as shown to result in longer and more masculine AGD since the AGD reflects the amount of androgens to which fetus is exposed [3].

The animal models have shown that, AGD shows a plasticity which can be mediated by local androgen/ estrogen effect [4]. Exposure to chemicals including dioxins exhibit anti androgenic activity and thus shorter AGD distance in rat models. Hence, AGD can be used as a marker of prenatal androgen activity [5]. The androgen exposure is often required for the prostate development also.

The lower urinary tract symptoms (LUTS) are due to bladder outlet obstruction (BOO) secondary to benign prostatic hyperplasia which poses a major problem for the existing medical care system. The patients with BPH usually suffer from the obstructive symptoms which are referred as lower urinary tract symptoms (LUTS) [6].

But the studies showing the relation between the anogenital distance and the lower urinary tract symptoms are scant. Hence this study was undertaken to ascertain the association between anogenital distance and benign prostatic hyperplasia related lower urinary tract symptoms.

## **Materials and methods**

A comparative study was undertaken in the Department of Urology of a tertiary care institution. About 50 patients with lower urinary tract symptoms due to benign prostatic hyperplasia and 50 controls without any

symptoms of lower urinary tract symptoms due to benign prostatic hyperplasia constituted the study sample. The clearance from institution ethics committee was obtained as per Helsinki declaration. An informed consent to participate in the study was obtained from all the subjects before including them in to the study. This study included cases with prostate weight up to 70 gms with history urinary retention, recurrent gross hematuria and recurrent UTI cases. Patients with history or direct rectal examination suggestive of malignancy, urethral stricture, those poor control of diabetes mellitus, neurological disorder and those who were on anticholinergic drugs,  $\alpha$  agonists or antidepressants were excluded from the study.

A detailed history of lower urinary tract symptoms (LUTS) was obtained from all the cases and controls. Symptoms described were evaluated on International Prostate Symptom score (IPSS), Prostate volume, Maximum flow rate ( $Q_{max}$ ), Prostate specific antigen (PSA), testosterone levels, height, weight were recorded. Two types anogenital distance was obtained AGDAP (from the centre of the anus to the cephalad insertion of penis) and AGDAS (from the center of the anus to the posterior base of the scrotum) were measured by using a digital calliper in lithotomy position with thighs at a distance of  $45^{\circ}$  angle to the examination table [7, 8]. Each AGD variant was measured three times and average was recorded. The data was obtained and analysed using Statistical Package for Social Services (SPSS vs 20).

## **Results**

The mean age of the LUTS group was 68.9 years and control was 69.3 years which was statistically not significant and hence the two groups were comparable. The mean BMI of the LUTS group was slightly higher in the LUTS group than the controls (**Table – 1**).

The prostate size was significantly smaller in controls when compared with the patients with LUTS. The PSA levels were significantly higher

in LUTS patients and also testosterone levels. The maximum flow rate was significantly higher in controls. The mean Anogenital distance (AP) was significantly lesser in LUTS group than the controls (Table – 2).

The BMI adjusted values of AGD (AS) also was significantly lower in LUTS group than the controls (Table – 3).

**Table – 1:** Comparison of baseline characteristics.

Baseline characteristics	LUTS group	Controls	T value	P value, Sig
Age (Mean ± SD)	68.9 ± 11.12	69.3 ± 10.36	0.186	0.853, NS
BMI (Kg/M <sup>2</sup> )	26.38 ± 3.87	25.27 ± 3.72	1.451	0.15, NS

**Table – 2:** Comparison of clinical findings of the study group.

Clinical findings	LUTS group	Controls	T value	P value, Sig
IPSS	24.84 ± 3.77	0.1 ± 0.3	46.244	0.000, Sig
Prostate volume (gms)	51.99 ± 6.88	33.35 ± 5.6	14.849	0.000, Sig
Prostate Specific Antigen (ng/mL)	3.48 ± 1.45	1.64 ± 1.36	6.539	0.000, Sig
Testosterone (ng/mL)	4 ± 1.6	6.02 ± 2.2	5.277	0.000, Sig
Q <sub>max</sub>	8.41 ± 2.05	17.51 ± 2.52	19.767	0.000, Sig
AGD <sup>AP</sup>	14.5 ± 2.49	13.22 ± 1.93	2.871	0.005, Sig
AGD <sup>AS</sup>	5.26 ± 1.52	5.82 ± 1.24	2.016	0.047, Sig

**Table – 3:** Comparison of adjusted AGD values.

Prostate findings	LUTS group	Controls	Adj P value, Sig
AGD <sup>AP</sup>	0.56 ± 0.14	0.53 ± 0.11	0.223, NS
AGD <sup>AS</sup>	0.21 ± 0.07	0.24 ± 0.06	0.033, Sig

## Discussion

This study was mainly undertaken compare the anogenital distance values in the patients with lower urinary tract symptoms due to benign prostatic hypertrophy when compared to the age matched controls. Many studies have been undertaken to correlate the prostate cancer and anogenital distance. But few studies are available to show the relationship between the anogenital distance and benign prostatic hyperplasia. The exact etiopathogenesis of benign prostatic hyperplasia is still unknown. But clinical data demonstrate that the androgen suppression and  $\alpha$  blockade can relieve the bladder outlet obstruction and increase the urinary flow rates in men with BPH [9].

Some parameters may help in prediction of development of BPH and BPH related LUTS. The literature available shows that, androgens can lead to the prostatic growth in the post

pubescent males [10]. Androgens are necessary for normal development of prostate [11, 12]. The studies also shown that, the AGD is a biological marker for prenatal androgen exposure which is sexually dimorphic features, longer in males than females [13].

A longer AGDAS is shown to be an indicator of more exposure to androgens may impair the proliferation of Leydig cells resulting in higher androgen levels in adulthood [14].

This study had shown longer AGD (AS & AP) in patients suffering with LUTS than the controls. A similar study had shown that, the AGD<sup>AS</sup> value of LUTS was significantly lower than the control group. There was no statistically significant difference between the two group in terms of mean adjusted ADG<sup>AP</sup> values. But mean adjusted AGD<sup>AS</sup> values were significantly lower in the LUTS group than the control group [15]. No

other studies were available to compare these results.

## Conclusion

Anogenital distance can be a marker for prevention of development of benign prostatic hyperplasia and BPH related LUTS. With this knowledge, preventive approaches including behavioural and dietary modifications can be applied to prevent the occurrence of LUTS.

## References

1. Sathyanarayana S, Beard L, Zhou C, Grady R. Measurement and correlates of anogenital distance in healthy, newborn infants. *Int J Androl.*, 2010; 33: 317-23.
2. Macleod DJ, Sharpe RM, Welsh M, Fiskens M, Scott HM, Hutchison GR, et al. Androgen action in the masculinization programming window and development of male reproductive organs. *Int J Androl.*, 2010; 33: 279– 87.
3. Hotchkiss AK, Parks-Saldutti LG, Ostby JS, Lambright C, Furr J, Vandenberg JG, et al. A mixture of the ‘anti-androgens’ linuron and butyl benzyl phthalate alters sexual differentiation of the male rat in a cumulative fashion. *Biol Reprod*, 2004; 71: 1852– 61.
4. Mitchell RT, Mungall W, McKinnell C, et al. Anogenital distance plasticity in adulthood: implications for its use as a biomarker of fetal androgen action. *Endocrinology*, 2015; 156: 24-31.
5. Gray LE Jr, Wilson VS, Stoker T, et al. Adverse effects of environmental anti-androgens and androgens on reproductive development in mammals. *Int J Androl.*, 2006; 29: 96-104.
6. McConell JD, Barry MJ, Bruskewitz RC, et al. Benign Prostatic Hyperplasia: Diagnosis and treatment. *Clinical Practice Guidelines*. No 8, AHCPR publication No. 94-0582. Rockville, Maryland, Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services, 1994.
7. Mendiola J, Stahlhut RW, Jørgensen N, et al. Shorter anogenital distance predicts poorer semen quality in young men in Rochester, New York. *Environ Health Perspect.*, 2011; 119: 958-63.
8. Parra MD, Mendiola J, Jørgensen N, et al. Anogenital distance and reproductive parameters in young men. *Andrologia*, 2016; 48: 3-10.
9. Lepor H. Pathophysiology of benign prostatic hyperplasia in the aging male population. *Rev Urol.*, 2005; 7 Suppl 4(Suppl 4): S3-S12.
10. Jarvis TR, Chughtai B, Kaplan SA. Testosterone and benign prostatic hyperplasia. *Asian J Androl.*, 2015; 17(2): 212-216.
11. Wilson JD. The Critical Role of Androgens in Prostate Development. *Endocrinol Metab Clin N Am.* 2011; 40: 577-90.
12. Yassin A, AlRumaihi K, Alzubaidi R, et al. Testosterone, testosterone therapy and prostate cancer. *Aging Male.*, 2019; 7: 1-9.
13. Kurzrock EA, Jegatheesan P, Cunha GR, Baskin LS. Urethral development in the fetal rabbit and induction of hypospadias: a model for human development. *J Urol.*, 2000; 164: 1786-92.
14. Wilson Jean D. Role of androgens in prostate development. *Endocrinol Metab Clin North Am.*, 2011; 40: 577-590.
15. Kutluhan MA, Sahin A, Urkmez A et al, The relationship between anogenital distance and benign prostatic hyperplasia related lower urinary tract symptoms. *Andrologia.*, 2020; 52: 7.