Section: General Medicine

Original Research Article

STUDY OF TESTOSTERONE LEVELS IN TYPE-II DIABETES MELLITUS MALE PATIENTS IN TELANGANA POPULATION

Syed Mustafa Ashraf[1], Syeda Asma Gulnaaz[2]

[1]Associate Professor, Department of General Medicine, Pratima Institute of Medical Sciences Kareem Nagar, Telangana, India.
[2]Assistant Professor, Department of Pharmacology, Deccan College of Medical sciences Hyderabad, Telangana, India.

Received : 05/05/2024
Received in revised form : 11/07/2024
Accepted : 26/07/2024

Keywords:
Chemiluminescene immune assay, HPLC, HbSA1C, androgens, Telangana.

Corresponding Author:
Dr. Syeda Asma Gulnaaz,
Email: gulnaaz86@gmail.com

DOI: 10.47009/jamp.2024.6.4.83

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2024; 6 (4); 415-417

Abstract

Background: Type-II DM is a major health problem globally; it affects mainly the cardio-vascular system, including the lipid profile, and also impairs hormone pathways like insulin and sex hormones in males, leading to impotency. **Materials and Methods:** 195 (one hundred ninety-five) type II DMs of different age groups were studied and compared with 190 normal (controlled) groups. The blood investigation included FBS and PP. Blood urea, serum creatinine, Hbs.A1C, lipid profile, urine albumin, creatinine ratio, and serum testosterone were estimated by chemiluminescence immune assay and HbA1c by HPLC. **Results:** The BMI, age, HBSA1C, and serum testosterone level were compared with the control group, and the p value was highly insignificant ($p<0.001$). **Conclusion:** It is confirmed that type II DM patients have lower serum testosterone levels. Diabetes mellitus affects the vascularity of vital organs and impairs the normal functions of tests, leading to male sterility. The present study helps physicians or endocrinologists treat such patients efficiently.

INTRODUCTION

Type-II DM is a metabolic disorder of multiple etiologies defined by chronic hyperglycemia with disturbances of carbohydrate, fat, and protein metabolism.[1] It results from defects in insulin secretion, insulin action (resistance), or both. It is reported that 171 million in 2000 will increase to 366 million in 2030, with a maximum increase in India.[2]

The etiology of diabetes is multifactorial and includes genetic factors, influenced by environmental factors, nutritional status such as obesity, sedentary life, or a stressful living atmosphere.[3] Diabetes mellitus is strongly associated with microvascular complications such as nephropathy, neuropathy, and retinopathy, leading to damage to vital organs; similarly, the vascularity of sominiferous tubules is impaired, and the release of testosterone is significantly decreased, leading to male sterility.[4] Hence, an attempt is made to evaluate the parameters of type-II DM and testosterone levels, and both parameters were correlated and compared in a healthy (controlled) group.

MATERIALS AND METHODS

195 males aged between 35-50 years with known type II diabetes mellitus regularly visited Pratima Institute of Medical Sciences, Kareem Nagar, Telangana 505417 were studied.

Inclusive Criteria: Type II DM patients, irrespective of the duration of diabetes, are currently on oral hypoglycemic drugs or insulin and gave their consent in writing for study was selected.

Exclusion Criteria: Patients aged less than 30 years with type II DM and patients with corticosteroids, testosterone, thyroid supplements, chronic renal disease, cirrhosis of the liver, and immune-compromised patients were excluded from the study.

Method: A detailed history, occupation, clinical examination, and investigation included CBC, fasting, and post-parandial blood sugar. Blood urea, serum creatinine $HBAS_1C$, lipid profile, urine albumin creatinine ratio, and diabetes mellitus were defined by ADA guidelines [5]. Serum testosterone levels (morning sample) were estimated using a chemiluminescence immunoassay. Low testosterone was defined as a serum testosterone level < 241 mg/dl, and the prevalence of its deficiencies was calculated. Estimation of HbS_1C (4.2–6.2%) performed by high-performance liquid

ISSN (O): 2687-5365; ISSN (P): 2753-6556

chromatography (HPLC) All important parameters, like age, BMI, mean $HBSA_1C$, and serum testosterone, were compared in healthy volunteers (controlled group).

The duration of the study was from May 2023 to June 2024.

Statistical Analysis: Various parameters in type II DM patients were studied and compared with a control group. The statistical analysis was carried out in SPSS software.

RESULTS

Table 1: Clinical manifestations in type-II DM patients – BMI 25.36 (± 2.24), $HbsA_1C$ 8.84 (± 1.92), serum testosterone 118.10 (± 83.4), age 55.13 (± 9.13)

Table 2: Distribution of type-II DM patients according to duration of disease

48 (24.6%) > 1 year, 69 (35.3%) 6–10 years, 53 (27.1%) 11–15 years, 25 (12.8%) > 15 years

Table 3: Comparison of clinical manifestations in type-II DM patients and a controlled group

- Age: 55.13 (± 9.13) in type-II DM, 37.76 (± 6.38) in the controlled group, t test was 21.5 and p<0.001
- BMI: 25.36 (± 2.24) in type-II DM, 24.84 (± 3.8) in the controlled group, t test was 1.6 and p > 0.10 (p value insignificant).
- $HBSA_1C$: 8.84 (± 1.92) in type-II DM, 4.78 (± 0.36) in the controlled group, t test was 28.6 and p<0.001.

Serum testosterone: 118.10 (± 83.4) in type-II DM, 405.8 (± 160.6) in the controlled group; the t test was 22.1 and p > 0.001.

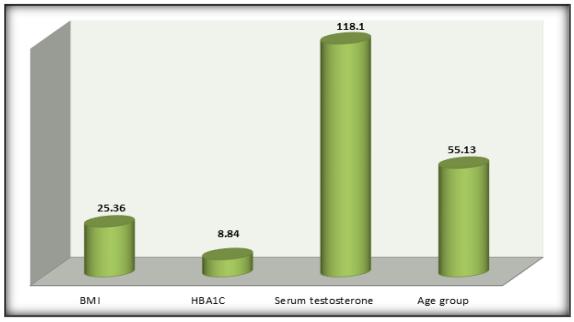

Figure 1: Clinical Manifestations in type-II DM patients

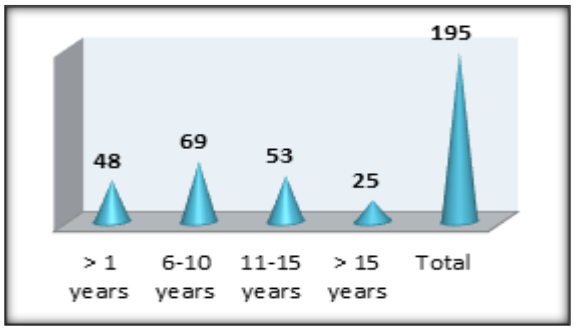

Figure 2: Distribution of type-II DM patients according to duration of disease

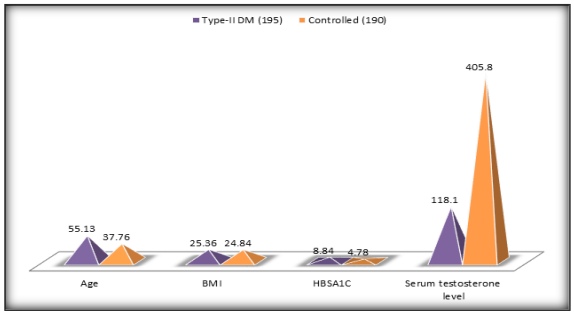

Figure 3: Comparison of clinical Manifestation in type-II DM patients with controlled groups

Table 1: Clinical Manifestations in type-II DM patients

(Total No. of patients: 195)

Manifestations	Mean ±SD
BMI	25.36 (± 2.24)
HBA_1C	8.84 (± 1.92)
Serum testosterone	118.10 (± 83.4)
Age group	55.13 (± 9.13)

Table 2: Distribution of type-II DM patients according to duration of disease

Duration of years	No. of patients (195)	Percentage
> 1 years	48	24.6
6-10 years	69	35.3
11-15 years	53	27.1
> 15 years	25	12.8
Total	195	100

Table 3: Comparison of clinical Manifestation in type-II DM patients with controlled groups

Parameter	Type-II DM (195)	Controlled (190)	t test	p value
Age	55.13 (± 9.13)	37.76 (± 6.38)	21.5	P<0.001
BMI	25.36 (± 2.24)	24.84 (± 3.8)	1.6	p>0.10
HBSA1C	8.84 (± 1.92)	4.78 (± 0.36)	28.6	P<0.001
Serum testosterone level	118.10 (± 83.4)	405.8 (± 160.6)	22.1	P<0.001

ISSN (O): 2687-5365; ISSN (P): 2753-6556

DISCUSSION

Present study of testosterone levels in type II DM diabetes male patients of Telangana. The clinical manifestations were 25.36 (± 2.24) BMI, HBSA1C level 8.84 (± 1.92), serum testosterone level was 118.10 (±83.4), and the age group was 55.13 (± 9.13) (Table 1). The distribution of type II DM patients was 48 (24.6%) > 1 year, 69 (35.3%) 6–10 years, 53 (27.1%) 11–15 years, and 25 (12.8%) > 15 years (Table 2). Comparison study of clinical manifestation with a health volunteer controlled group Age, HBSA1C, and serum testosterone level had a significant p value ($p<0.001$) (Table 3). These findings are more or less in agreement with previous studies.[5,6,7]

Defining the lower limit of normal for S. testosterone levels poses a challenge for physicians. The adverse clinical outcomes that occur in type II DM are not known.[8] Testosterone in men is synthesized and secreted into circulation almost exclusively by the cells of the leydig of the testes. It is mostly bound to plasma proteins. S. testosterone is composed of 0.5 to 3% of free testosterone unbound to plasma proteins, 30–44% sex hormone-binding globulin (SHBG)-bound testosterone, and 54-60% albumin-bound testosterone.[9] Moreover, variations in S. testosterone metabolism are associated with environmental and/or genetic factors.[10]

It was experimented on in lower animals (mouse) that testosterone therapy increased muscle mass and reduced fat mass, both of which were expected to decrease insulin resistance. It was also observed in mice that testosterone regulated skeletal muscle genes involved in glucose metabolism, which led to decreased systemic insulin resistance.[11]

It can be hypothesized that a low S. testosterone level could contribute to the development of obesity and type II DM through changes in body composition. In obese men, the peripheral conversion from testosterone to estrogen could attenuate the amplitude of luteinizing hormone pulses and centrally inhibit testosterone production.[12] Moreover, leptin and adipokine have been shown to be inversely correlated with serum testosterone levels in men.

Low testosterone levels can be perpetuated through defects in the HPG axis. Hence, type II DM patients had hypogonatropic hypogonadism. Ageing is also well known to result in a decline in sex hormone levels and is likely a combination of testosterone and pituitary hypothalamic defects. In elderly men, there is a reduced testicular response to gonadotrophins with suppressed and altered pulsality of the hypothalamic pulse generation.

Low testosterone is commonly associated with a high prevalence of metabolic risk factors, including insulin resistance, hypertension, dyslipidemia, obesity (particularly central adiposity), CVD, and type II DM, because testosterone has been shown to dilate coronary vessels in animals and men, suggesting that it might be an important regulator of vasculature compliance and a modifier of blood pressure.

CONCLUSION

The present study of serum testosterone levels in type II DM patients's causes insulin resistance, obesity, vascular dysfunction, and inflammation. There is a higher prevalence of type II DM patients across the world. This study demands further study of genetic, hormonal, nutritional, and pharmacological factors to clarify whether low testosterone is merely a reflection of poor cardio-vascular risk factor control or is really causing adverse clinical outcomes or higher viscosity of blood in type II DM patients may prevent or retard the flow of testosterone, which leads to low testosterone hormone is still unclear.

Limitation of study: Due to the tertiary location of the research center, the small number of patients, and the lack of the latest techniques, we have limited findings and results.

- This research work has been approved by the ethical committee of the Pratima Institute of Medical Sciences, Kareem Nagar, Telangana 505417
- No Conflict of Interest
- Self-Funding

REFERENCES

1. Fakui M, Kitgawa Y.: Association between serum testosterone concentration and carotid atherosclerosis in men with type 2 diabetes. Diabetes Care 2003, 26; 1869–1873.
2. Singh R, Artoza JN: Androgens stimulate myogenic differentiation and inhibit adiposeness in the C3H 10T1/2 androgen receptor-mediated pathway. Endocrinology 2003, 144: 5081–5088.
3. Corona G, Mannucci E: Associate of hypogonadism and type II diabetes in men attending our patients erectile dysfunction clinic Int. J. Impot Res. Rev. 2006, 18; 190–7.
4. Marcus GJ, Dunford RA: Simple linked immune assay for testosterone and steroids, 1985, 46; 975-86
5. Lott JA, Turner K: Evaluation of the Trinders glucose oxidase method for measuring glucose in serum and urine, Clin. Chem. 1975, 21 (1): 1754–60.
6. Xue B, Less A: Protein tyrosine phosphatase 1B deficiency reduces insulin resistance and the diabetic phenotype in mice with polygenic insulin resistance. The Journal of Biological Chemistry 2007, 282 (33); 23829–40.
7. Schean AJ, Pathophysiology of Type II Diabetes, Acta Clinica Belgica, 2004, 58(6), 336-41.
8. Taish AM, Saad F, and Feely RJ: The Dark Side of Testosterone Deficiency, 111. Cardio-vascular Disease J. Androl 2009, 30; 477–494.
9. Bhasin S, Cunningham GR: Testosterone therapy in men with androgen deficiency synchronies J. Clin. Endocrinal Metab 2010, 95; 2536–2559.
10. Janes TH, Arver S: Testosterone replacement in hypogonadal men with type II DM and/or metabolic syndrome, Diabetes Care 2011, 4: 828–837.
11. Kalyani RR, Dobs AS: Androgen deficiency, diabetes, and the metabolic syndrome in men. Curr. Opin. Endocrinal. Diabetes Obes. 2007, 226-34.
12. Dunn JF, Nisula BC: Transport of steroid hormones binding 21 endogenous steroids to both testosterone-binding globulin and human plasma J. Clin. Endocrinal Metabolics 1981, 53; 58–68.

ISSN (O): 2687-5365; ISSN (P): 2753-6556