

## Study of Testosterone Levels in Type-II Diabetes Mellitus Male Patients in Telangana Population

Mohammed Asif Muzaffer Iqubal<sup>1</sup>, Mohammed Mudassir Ali<sup>2</sup>, Md Muneer Ahmed<sup>3</sup>, Juwairia Mohammed Fariduddin<sup>4</sup>

<sup>1</sup>Assistant Professor, Department of General Medicine, Deccan College of Medical Sciences, Kanchanbagh, Hyderabad, Telangana-500002

<sup>2</sup>Associate Professor, Department of General Medicine, Deccan College of Medical Sciences, Kanchanbagh, Hyderabad, Telangana-500002

<sup>3</sup>Assistant Professor, Department of General Medicine, Deccan College of Medical Sciences, Kanchanbagh, Hyderabad, Telangana-500002

<sup>4</sup>Consultant Biochemist Fernandez Hospital Hyderguda, Hyderabad

Received: 25-07-2023 / Revised: 28-08-2023 / Accepted: 30-09-2023

Corresponding author: Dr. Juwairia Mohammed Fariduddin

Conflict of interest: Nil

### Abstract:

**Background:** Type-II DM affects millions of people in India and globally, but the association between serum testosterone levels (STL) and type-II DM is still unclear, but it impairs reproductive health and quality of life.

**Method:** 95 (ninety-five) type II DM patients of different age groups were studied and compared with 90 normal (controlled) groups. The blood investigation included FBS, PP Blood sugar, Blood urea, serum creatinine, HBA<sub>1c</sub>, lipid profile, urine albumin, creatinine ratio, serum testosterone were estimated by chemiluminescence immune assay and HbA<sub>1c</sub> by HPLC.

**Results:** The BMI, age, HBA<sup>1</sup>C, and serum testosterone level were compared with the control group, and the p value was highly insignificant (p<0.001).

**Conclusion:** The present pragmatic study has confirmed that type II DM patients have significantly lower testosterone and higher sugar level was proved.

**Keywords:** Chemiluminescence Immune Assay, HPLC, HBA<sup>1</sup>C, Androgens, Telangana.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

### Introduction

Type-II diabetes mellitus (DM) affects 285 million people globally. This number is expected to reach 438 million by the year 2030. Asian countries populations develop diabetes at a younger age and obesity and weight gain [1]. The association between low serum testosterone and DM has recently received substantial attention. The reduced total testosterone levels have been associated with insulin resistance and a subsequent risk of developing type II DM [2]. The main symptoms of low serum testosterone (LST) are reduced libido and erectile dysfunction, reduced muscle mass, depressed mood, fatigue, low energy, and impaired quality of life [3]. It is reported that the potential metabolic consequences of age-associated metabolic changes such as abdominal obesity, diabetes, and markers of pre-diabetics Hypogonadism (HG) is a clinical condition consisting of both symptoms and biochemical signs of testosterone deficiency. Usually, total testosterone concentrations (levels) are determined, to a large extent, by the circulating sex hormone-binding globulin (SHG) concentration. In the blood

of normal men, 44% of total testosterone is bound to SHBG. 2% is unbound (free testosterone) (FT), and 54% circulates bound to albumin and other proteins [4]. It is yet to be known whether the lower testosterone levels in diabetes are associated with changes in luteinizing hormone (LH) and follicular stimulating hormone (FSH). It was also confirmed that diabetic neuropathy patients had low testosterone, LH, and FSH levels.

The prevalence of serum testosterone (LST) is largely unknown to many patients in India and western countries as well. Hence, an attempt was made to evaluate testosterone levels in type II DM male patients with chronic and newly developed diabetes.

### Material and Method

95 males aged between 30-50 years known diabetes mellitus regularly visited Deccan College of Medical Sciences, Kanchanbagh – Hyderabad, Telangana were studied.

### Inclusive Criteria

Type II DM patients, irrespective of the duration of diabetes, are currently on oral hypoglycemic drugs or insulin.

### Exclusion Criteria

Patients age 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 HBA1C, lipid profile, urine for 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 HBA1C (4.2–6.2%) performed by high-performance liquid chromatography (HPLC) All important parameters, like age, BMI, mean HBA1C, and serum testosterone, were compared in healthy volunteers (controlled group).

The duration of the study was from May 2022 to June 2023.

### 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.

### Observation and Results

Table 1: Clinical manifestation in type-II DM

Age – 55.14 ( $\pm 9.14$ ), BMI 25.38 ( $\pm 2.25$ ), HBA<sub>1C</sub> 8.82 ( $\pm 1.90$ ), serum testosterone 119.11 ( $\pm 84.2$ )

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

23 (24.2%) > 6 years, 34 (35.7%) 6-10 years, 26 (27.3%) 11-15 years, 12 (12.6%) > 15 years

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

- Age – 55.14 ( $\pm 9.14$ ) in type-II DM patients, 38 ( $\pm 6.40$ ) in controlled group, t test was 14.08 and  $p < 0.001$ .
- BMI – 25.38 ( $\pm 2.23$ ) in type-II DM patients, 24.86 ( $\pm 3.10$ ) in controlled group, t test was 1.37 and  $p > 0.17$  (p value insignificant) .

- HBA<sub>1C</sub> – 8.82 ( $\pm 1.90$ ) in type-II DM patients, 4.78 ( $\pm 0.38$ ) in controlled group, t test was 20.3 and  $p < 0.001$ .
- Serum testosterone – 119.11 ( $\pm 84.2$ ) in type-II DM patients, 405.8 ( $\pm 168.6$ ) in controlled group, t test was 14.5 and  $p > 0.001$ .

### Discussion

Present study of testosterone levels in type II DM diabetes mellitus male patients in the Telangana population. The clinical manifestations were age 55.14 ( $\pm 9.14$ ), BMI 25.38 ( $\pm 2.25$ ), HBA<sub>1C</sub> 8.82 ( $\pm 1.90$ ), and serum testosterone 119 ( $\pm 84.2$ ) (Table 1). The distribution of type II DM patients according to duration of disease 23 (24.2%) > 6 years, 34 (35.7%) 6–10 years, 26 (27.3%) 11–15 years, 12 (12.6%) > 15 years (Table 2), The comparison of clinical manifestation in type-II DM patients and controlled group: age 55.14 ( $\pm 9.14$ ) in type-I DM, 38.77 ( $\pm 6.40$ ) in controlled group, t test was 14.08 and  $p < 0.001$ , BMI 25.38 ( $\pm 2.25$ ) in type-II DM group, 24.86 ( $\pm 3.10$ ) in controlled group, t test 1.37 and  $p > 0.17$  (p value is insignificant) HBA<sub>1C</sub> 8.82 ( $\pm 1.90$ ) in type-II DM, 4.78 ( $\pm 0.38$ ) in controlled group, t test was 20.3 and  $p < 0.001$ , Serum testosterone 119.11 ( $\pm 84.2$ ) in type-II DM, 405.8 ( $\pm 168.6$ ) in controlled group, t test 14.5 and  $p < 0.001$  (Table 3). These findings are more or less in agreement with previous studies [6,7,8].

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 [9]. Testosterone in men is synthesised 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 [10]. Moreover, variations in S. testosterone metabolism are associated with environmental and/or genetic factors [11].

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 [12].

It can be hypothesised 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 oestrogen could attenuate the amplitude of luteinizing hormone pulses and centrally inhibit testosterone production.

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 pulsatility 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.

**Summary and Conclusion**

The present study of serum testosterone levels in type II DM patient’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 cardiovascular 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 un-clear.

**Limitation of study**

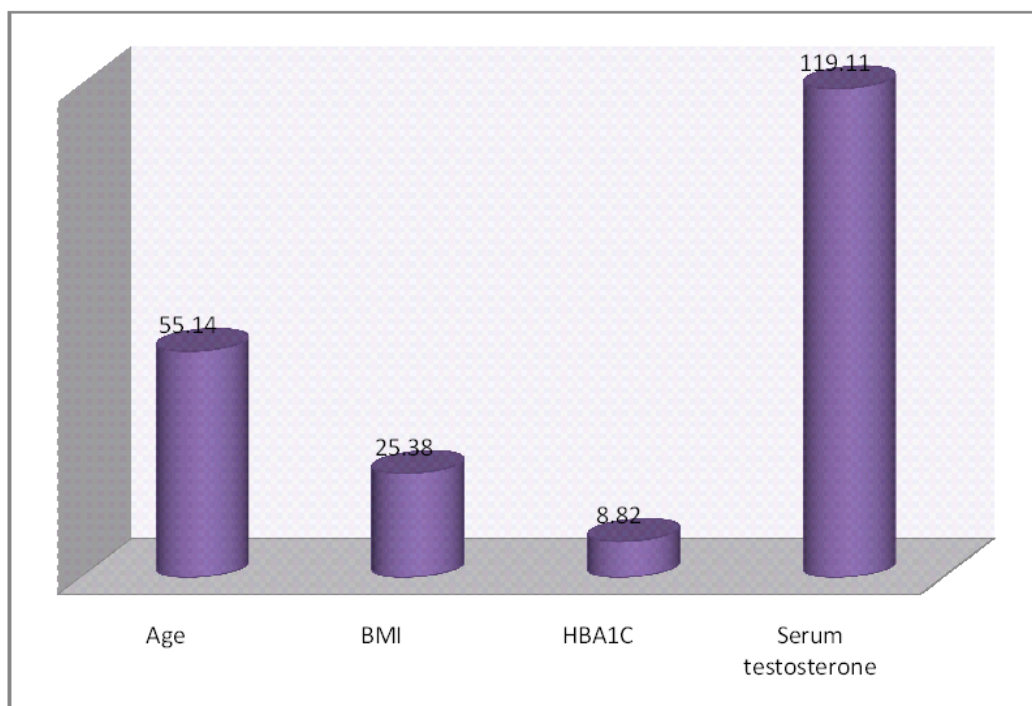
Due to tertiary location of research centre, small number of patients and lack of latest techniques, we have limited findings and results.

- This research paper is approved by Ethical committee of Deccan College of Medical Sciences, Kanchanbagh – Hyderabad, Telangana-500002
- No Conflict of Interest
- Self Funding

**Table 1: Clinical Manifestations in type-II DM patients**

Manifestations	Mean ±SD
Age	55.14 (± 9.14)
BMI	25.38 (± 2.25)
HBA <sub>1</sub> C	8.82 (± 1.90)
Serum testosterone	119.11 (± 84.2)

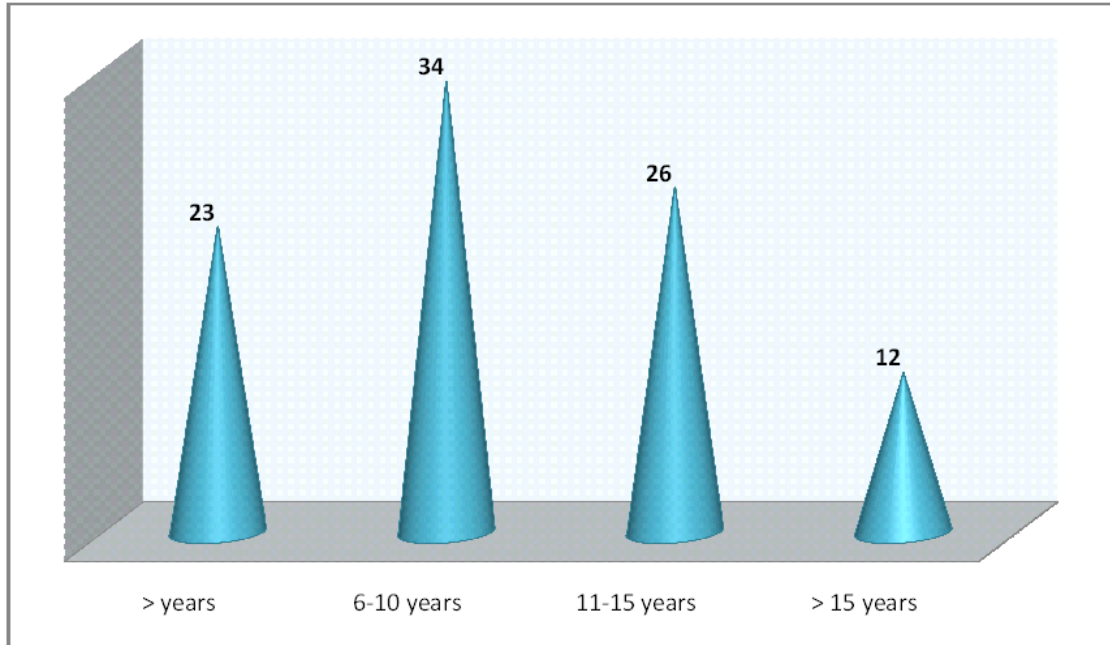
(Total No. of patients: 95)



**Table 1: Clinical Manifestations in type-II DM patients**

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

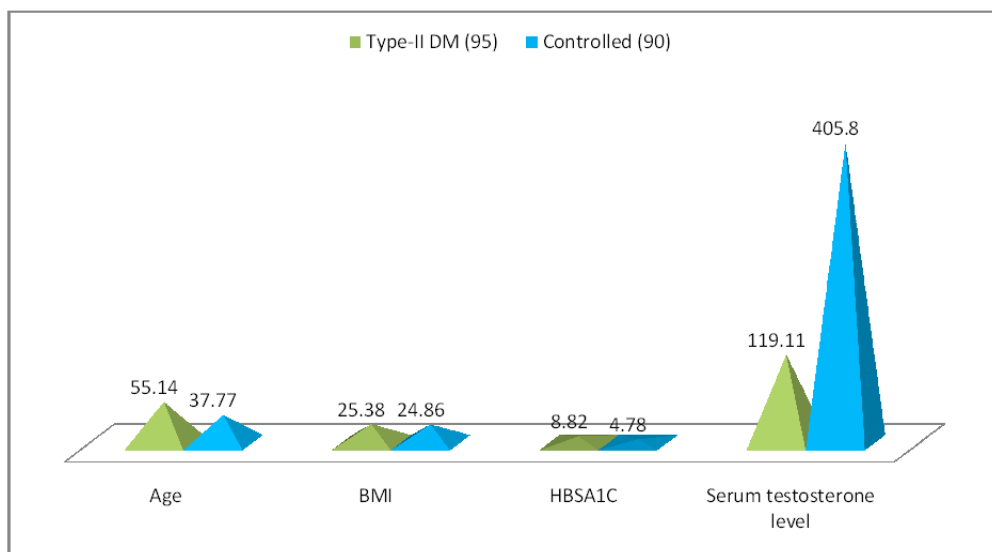
Duration of years	No. of patients (95)	Percentage
> years	23	24.2
6-10 years	34	35.7
11-15 years	26	27.3
> 15 years	12	12.6



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

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

Parameter	Type-II DM (95)	Controlled (90)	t test	p value
Age	55.14 (± 9.14)	37.77 (± 6.40)	14.08	P<0.001
BMI	25.38 (± 2.25)	24.86 (± 3.10)	1.37	p>0.17
HBA <sub>1</sub> C	8.82 (± 1.90)	4.78 (± 0.38)	20.3	P<0.001
Serum testosterone level	119.11 (± 84.2)	405.8 (± 168.6)	14.5	P<0.001



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

**References**

1. 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.
2. Kalyani RR, Dobs AS – Androgen deficiency, diabetes, and the metabolic syndrome in men. Curr. Opin. Endocrinal. Diabetes Obes. 2007; 226-34.
3. Janes TH, Arver S – Testosterone replacement in hypogonadal men with type II DM and/or metabolic syndrome, Diabetes Care 2011; 4: 828–837.
4. Bhasin S, Cunningham GR Testosterone therapy in men with androgen deficiency synchronies J. Clin. Endocrinal Metab 2010; 95; 2536–2559.
5. Taish AM, Saad F, Feely RJ – The Dark Side of Testosterone Deficiency 111. Cardiovascular Disease J. Androl 2009; 30; 477–494.
6. Schean AJ Pathophysiology of Type II Diabetes, Acta Clinica Belgica, 2004;58(6), 336-41.
7. 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.
8. Lott JA, Turner K – Evaluation of Trinders glucose oxidase method for measuring glucose in serum and urine, Clin. Chem. 1975; 21 (1): 1754–60.
9. Marcus GJ, Dunford RA – Simple linked immune assay for testosterone and steroids, 1985; 46; 975-86
10. 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.
11. 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.
12. Fakui M, Kitgawa Y – Association between serum testosterone concentration and carotid atherosclerosis in men with type 2 diabetes. Diabetes Care 2003; 26; 1869–1873.