

LOGISTIC REGRESSION ANALYSIS FOR ERECTILE DYSFUNCTION AND LOSS OF LIBIDO WITH SEX HORMONE LEVEL CHANGES IN MEN

Jayaprakash Narayanan¹, Ezhil Sundar V², Kirankumar J³

Received : 07/10/2025
Received in revised form : 21/11/2025
Accepted : 10/12/2025

Keywords:

Erectile dysfunction, libido, sex hormones, logistic regression, prolactin, follicle-stimulating hormone.

Corresponding Author:

Dr. Kirankumar J,
Email: kumar.jkkdr@gmail.com

DOI: 10.47009/jamp.2025.7.6.189

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

Int J Acad Med Pharm
2025; 7 (6); 717-721



¹Associate Professor, Department of Urology, K.A.P. Viswanatham Government Medical College, Tiruchirappalli, Tamilnadu, India.

²Associate Professor, Department of Urology, Madras Medical College & RGGGH, Chennai, Tamilnadu, India.

³Senior Resident, Department of Urology, Madras Medical College & RGGGH, Chennai, Tamilnadu, India.

ABSTRACT

Erectile dysfunction (ED), previously known as impotence, refers to the consistent inability to attain or sustain an erection adequate for satisfactory sexual activity. A decline in sexual desire, termed loss of libido, indicates reduced interest or drive for sexual engagement, which can be transient or persistent. While it is common and variable, persistent issues may warrant medical evaluation due to potential personal and relational impacts.

This study aims to apply logistic regression analysis to assess the association between ED—with or without loss of libido—and changes in male sex hormone levels. Conducted in 2022 at Q Med Hospital, Thiruchy, Tamil Nadu, the research involved male patients presenting with ED and/or reduced libido. Hormonal profiles including total testosterone, prolactin, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and estradiol were measured and categorized across age groups and symptom subgroups.

Key findings highlight significant alterations in prolactin and FSH levels. Among 53 participants, 41 exhibited hyperprolactinemia in association with ED, while 17 showed elevated prolactin linked with loss of libido. None had hypoprolactinemia. Nineteen participants reported loss of libido. Logistic regression analysis revealed that prolactin and FSH levels were statistically significant predictors, with odds ratios of 0.102 and 0.043, respectively, each with one degree of freedom.

INTRODUCTION

Erectile dysfunction (ED) is defined as the continuous inability to achieve or maintain an erection adequate for satisfactory sexual intercourse. It significantly impacts men's quality of life, emotional well-being, self-esteem, and interpersonal relationships, and may also contribute to psychological distress.^[1,2] The Massachusetts Male Aging Study estimates the prevalence of ED to be around 52% among men aged 40 to 70 years.^[3] Due to social stigma and embarrassment, more than 70% of ED cases often remain undiagnosed.^[4] Importantly, ED can also serve as an early indicator of underlying systemic conditions requiring medical attention, prompting comprehensive screening by urologists. Loss of libido, or diminished sexual desire, can either be short-lived or chronic. While fluctuations in sex drive are normal, persistent low libido can interfere with relationships and emotional health, warranting clinical evaluation.

Testosterone plays a pivotal role in regulating male sexual behavior by initiating sexual desire and promoting masculine characteristics.^[5] It is secreted in a pulsatile manner by Leydig cells of the testes under the influence of luteinizing hormone, typically showing a diurnal variation with peak levels in the early morning. Free testosterone, the biologically active component, constitutes about 2% of the total testosterone, with the rest bound to plasma proteins such as albumin and sex hormone-binding globulin.^[6] The physiologically active form enters target cells, where it can be converted to dihydrotestosterone by 5-alpha reductase. Alterations in plasma protein levels can therefore affect the availability of free testosterone. Low testosterone levels can reduce smooth muscle relaxation in the corpus cavernosum, contributing to ED.^[7]

Prolactin, a polypeptide hormone secreted by the anterior pituitary, is primarily regulated by dopamine from the hypothalamus. Its production can also be influenced by factors like stress, nipple stimulation, light exposure, and olfactory stimuli. TRH and

dopamine antagonists, such as certain antipsychotics, can increase prolactin levels. Although prolactin levels are typically low in males, elevated levels (hyperprolactinemia) may indicate underlying conditions such as pituitary tumors or drug side effects. In men, hyperprolactinemia is often associated with reduced libido and erectile dysfunction, likely due to suppression of gonadotropin-releasing hormone (GnRH) and subsequent hormonal imbalance.^[8,9]

Given the complex interplay between hormonal levels and sexual function, a comprehensive evaluation of sex hormones is essential in the assessment of ED. Despite cost and time constraints often limiting extensive investigations, it remains crucial to identify treatable endocrine causes.^[10] This study explores the relationship between sex hormone profiles and ED, with or without loss of libido, in affected men.

Participants were included if they were 18 years or older, had a clinical diagnosis of ED for at least six months, and reported regular sexual activity with a female partner. Exclusion criteria included history of pelvic surgery or radiation, chronic neurological conditions, hypogonadism, excessive alcohol intake, and current medication use. Each participant completed a detailed questionnaire assessing ED-related concerns. A physical examination was followed by relevant hormone testing.^[11,12]

After fasting for 10 hours, blood samples were collected at 15-minute intervals between 8:00 and 10:00 a.m.^[13] Hormone levels assessed included total testosterone, prolactin, FSH, LH, and estradiol, using chemiluminescent immunoassay (ACS:180-Chiron) and competitive radioimmunoassay (DSL) techniques. FSH and LH levels help determine whether hypogonadism is of primary or secondary origin.

MATERIALS AND METHODS

This study was conducted at the outpatient department of Q Med Hospital, located in Thiruchy, Tamil Nadu, over a one-year period from January 1 to December 31, 2022. The research included male patients who presented with erectile dysfunction, either with or without associated loss of libido. Each participant underwent a comprehensive evaluation of sex hormone levels.

The hormone panel included total testosterone, prolactin, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and estradiol. These values were stratified by age group and by the presence or absence of libido loss in patients with erectile dysfunction. All hormonal assays were conducted using standardized laboratory techniques to ensure consistency and reliability of data.

For the statistical evaluation, logistic regression analysis was employed to assess the association between hormone levels and the occurrence of ED and/or libido loss. The model adopted was based on

principles outlined by Douglas C. Montgomery and colleagues (2006), designed for binary outcomes.

Let Y represent the binary response variable (e.g., presence or absence of loss of libido), taking values of 1 (event occurs) or 0 (event does not occur). The odds of the event occurring are expressed as:

$$\text{Odds } (Y=1) = P / (1 - P)$$

Where P is the probability of the event. Taking the natural logarithm of the odds gives the logistic regression equation:

$$\text{Log } (P / (1 - P)) = \beta_0 + \beta_1 X$$

Where β_0 is the intercept, and β_1 is the coefficient for the predictor variable X .

This model was applied to assess the influence of various sex hormone levels on the presence of erectile dysfunction with or without loss of libido, with the primary focus on prolactin and follicle-stimulating hormone levels.^[14]

RESULTS AND DISCUSSION

A total of 53 male participants presented with erectile dysfunction. (Table 2) Age-wise classification revealed that 34 patients were below 39 years (young adults), 16 were between 40 and 59 years (middle-aged), and 3 were over 60 years (older adults).

Among these patients, 41 exhibited elevated prolactin levels (hyperprolactinemia), while 12 had prolactin values within the normal range. No cases of low prolactin (hypoprolactinemia) were recorded. [Table 3] Nineteen individuals reported loss of libido, while 34 did not experience reduced sexual desire. [Table 4]

The majority of patients with hyperprolactinemia (27 out of 41) were in the younger age category. Again, hypoprolactinemia was absent in all age brackets. [Table 5]

Seventeen of the 19 patients with loss of libido were also found to be hyperprolactinemic. Only two individuals with libido loss had normal prolactin levels, and none had decreased prolactin. [Table 6]

In assessing the model fit, the Hosmer–Lemeshow test for loss of libido yielded a chi-square significance value of 0.223. Since this value is greater than 0.05, the logistic regression model was considered an appropriate fit for the observed dataset. [Table 7]

Further evaluation using the classification table showed a correct classification rate of 77.4% for predicting loss of libido. This indicates a good predictive performance of the model. [Table 8]

In the logistic regression model, the dependent variable was loss of libido, while the independent variables included age, comorbidities, alcohol use, history of trauma, smoking status, and hormone levels (FSH, LH, prolactin, testosterone, and estradiol). [Table 9]

Among all predictors, prolactin and FSH emerged as statistically significant variables influencing libido. Prolactin had an odds ratio of 0.102, indicating a strong inverse relationship with libido. FSH also

showed a significant negative association, with an odds ratio of 0.043. [Table 9] These findings suggest that elevated levels of prolactin and FSH may

contribute to sexual dysfunction, particularly in reducing libido among men already affected by erectile dysfunction.

Table 1: Erectile Dysfunction Group with or Without Loss of Libido

Patient ID	Age	Co-Morbid	Alcoholic	Trauma	Smoker	Follicle Stimulating Hormone	Luteinising Hormone	Prolactin	Testosterone	Estradiol	Loss of Libido
P1	31	Nil	No	No	Yes	3.84	2.94	13.59	945	<90	No
P2	37	Nil	No	No	No	5.16	12.5	35.4	641	<90	No
P3	37	Nil	No	No	No	14.2	5.57	22.85	309.5	<90	Yes
P4	22	Nil	No	No	No	2.19	7.06	25.71	281.8	<90	Yes
P5	30	Nil	No	No	No	13.1	6.25	19.88	779.7	<90	Yes
P6	26	Nil	Yes	No	No	2.09	6.42	15.2	590.6	<90	Yes
P7	34	Nil	Yes	No	Yes	1.47	7.67	24.2	322.8	<90	No
P8	26	Nil	No	Yes	No	1.56	3.71	19.5	353.8	<90	No
P9	32	Nil	No	Yes	No	5.6	6.5	23.7	261.7	<90	No
P10	40	Nil	Yes	No	Yes	3.42	2.31	12.85	216.7	<90	Yes
P11	39	Nil	No	No	No	2.49	3.24	27	215.8	<90	No
P12	37	Nil	No	No	No	5.33	4.05	41.2	88.7	<90	No
P13	33	Nil	Yes	No	No	8.5	6.65	18.73	457.2	210	Yes
P14	38	Nil	Yes	No	No	4.69	3.76	19.12	585.8	<90	Yes
P15	30	Nil	Yes	Yes	No	6.6	9.38	26	211.3	<90	No
P16	34	Dm	Yes	Yes	No	4.79	10.5	19.5	281.9	<90	No
P17	25	Nil	Yes	Yes	No	< 1	3.97	21.7	276	<90	No
P18	45	Nil	No	No	No	2.87	5.18	28.2	160.5	<90	No
P19	28	Nil	No	No	No	< 1	2.29	28.56	235	643.8	No
P20	32	Nil	No	No	No	3.85	8.5	40	352.5	<90	Yes
P21	48	Nil	No	No	No	6.4	12.5	31.61	188.5	<90	Yes
P22	43	Nil	Yes	No	No	10.2	3.8	8.8	705.2	<90	No
P23	34	Sht	No	No	Yes	7.5	9.8	15.81	354	<90	No
P24	23	Nil	No	No	No	<1	11.2	21.36	211.3	<90	No
P25	60	Dm	Yes	No	No	11.5	9.5	9.56	429.2	<90	No
P26	51	Sht	Yes	No	No	4.8	6.4	5	514.4	<90	No
P27	47	Copd	No	No	No	12.5	8.7	18.25	367.3	<90	No
P28	35	Nil	No	Yes	No	1.1	11.2	19.5	296	<90	No
P29	40	Nil	Yes	No	No	4.5	10.9	32.28	202.5	<90	Yes
P30	29	Nil	Yes	No	No	6.8	7.9	23.5	365.2	<90	Yes
P31	45	Sht	Yes	No	Yes	10.5	11.4	24.6	288.3	<90	No
P32	26	Nil	No	No	No	4.9	8.1	19	378.4	<90	No
P33	45	Nil	Yes	Yes	No	9.4	12.7	21	245.4	<90	No
P34	25	Nil	Yes	No	No	2.6	6.4	12.5	501.4	<90	No
P35	35	Sht	No	No	No	13.5	10.3	30	180.4	<90	Yes
P36	62	Nil	No	No	No	5.7	7.1	9.25	544.5	<90	No
P37	45	Dm	No	No	No	9.4	8.2	19.5	398.3	<90	No
P38	25	Nil	No	No	No	11.2	13.5	20.7	279.3	<90	Yes
P39	56	Dm	Yes	Yes	Yes	15.3	10.7	29.35	160.4	<90	Yes
P40	24	Nil	No	No	No	<1	6.4	12.36	411.9	<90	No
P41	36	Nil	No	No	No	2.7	12.5	27	209.6	<90	No
P42	34	Nil	Yes	No	No	12.6	8.7	19.8	491.3	<90	No
P43	28	Nil	No	No	No	8.2	11.9	28.3	201.4	<90	Yes
P44	37	Nil	Yes	No	No	8.2	8.4	20.1	541.2	<90	Yes
P45	53	Sht	Yes	Yes	No	10.3	7.9	20.65	396.3	<90	No
P46	60	Dm	No	No	No	6.4	10.7	28	432.6	<90	No
P47	27	Nil	No	No	Yes	8.2	9.7	8.46	625.2	<90	No
P48	42	Nil	No	No	No	5.9	13.2	21	356.9	<90	No
P49	34	Nil	No	No	No	1.9	11.1	21.4	198.3	519.1	Yes
P50	50	Copd	Yes	No	No	4.5	9.2	18.5	305.9	<90	No
P51	31	Nil	Yes	No	No	3.9	10.9	9.25	518.7	<90	No
P52	49	Nil	No	No	No	11.7	9.5	23.65	260.5	<90	Yes
P53	45	Dm	No	Yes	No	8.7	4.8	21.14	290.1	<90	Yes

Table 2: Frequency of Age Group

Age	Frequency	Percentage
Below 39 (Young Age Adults)	34	64.2
40 - 59 (Middle Age Adults)	16	30.2
Above 60 (Old Age Adults)	3	5.7
Total	53	100.0

Table 3: Frequency of Prolactin Level

Prolactine Level	Frequency	Percentage
Hypoprolactineamic	0	0
Normal Prolactin	12	22.6
Hyperprolactineamic	41	77.4
Total	53	100.0

Table 4: Frequency of Loss of Libido

Loss of Libido	Frequency	Percentage
Yes	19	35.8
No	34	64.2
Total	53	100.0

Table 5: Cross Table for Prolactine Level and Age Group

Prolactin	Age			Total
	Young	Middle	Old	
Hypoprolactineamic	0	0	0	0
Normal Prolactin	7	3	2	12
Hyperprolactineamic	27	13	1	41
Total	34	16	3	53

Table 6: Cross Table for Prolactine Level and loss of Libido

Prolactin	Loss of Libido		Total
	Yes	No	
Hypoprolactineamic	0	0	0
Normal Prolactin	2	10	12
Hyper Prolactineamic	17	24	41
Total	19	34	53

Table 7: Hosmer and Lemeshow Test for Loss of Libido

Test	Chi-square	df	Sig.
Observed and expected frequency of loss of libido	10.643	8	0.223

Table 8: Observed and Predicted 2x2 Contingency Table for Loss of Libido

Observed		Predicted		
		Loss of Libido		Percentage Correct
		Yes	No	
Loss of Libido	Yes	11	8	57.9
	No	4	30	88.2
Overall Percentage				77.4

Table 9: Contribution of Each Independent Variables for Loss of Libido

Independent Variables	Estimated Value B	S.E.	Wald	Df	Sig.	Exp(B)
Age	-0.289	0.711	0.166	1	0.684	0.749
Co-Morbid	1.375	0.733	3.519	1	0.061	3.955
Alcoholic	0.895	0.823	1.183	1	0.277	2.447
Trauma	-1.301	1.061	1.505	1	0.220	0.272
Smoker	0.745	1.147	0.422	1	0.516	2.106
Follicle Stimulating Hormone	-3.147	1.252	6.324	1	0.012	0.043
Luteinising Hormone	0.788	0.817	0.931	1	0.335	2.199
Prolactin	-2.285	1.245	3.369	1	0.046	0.102
Testosterone	-0.920	1.232	0.557	1	0.455	0.399
Estradiol	-1.981	1.758	1.270	1	0.260	0.138
Constant	13.514	6.283	4.626	1	0.031	7.395

CONCLUSION

This study analyzed the relationship between sex hormone levels and erectile dysfunction, with or without accompanying loss of libido, using logistic regression. The results revealed that significant hormonal imbalances, particularly involving prolactin and follicle-stimulating hormone (FSH), were commonly observed among affected individuals.

Of the 53 participants, 41 had elevated prolactin levels in association with erectile dysfunction, while

12 had normal prolactin values. None exhibited low prolactin levels. Among those reporting loss of libido, 17 were hyperprolactinemic and 2 had normal prolactin levels. A total of 19 patients (35.8%) experienced reduced sexual desire.

Logistic regression analysis identified prolactin and FSH as significant predictors for loss of libido. The odds ratios for these hormones were 0.102 and 0.043, respectively—each with one degree of freedom—indicating a strong inverse relationship between these hormone levels and libido in men with ED.

These findings underscore the importance of hormonal evaluation in the clinical assessment of men presenting with erectile dysfunction and decreased libido. Identifying endocrine abnormalities such as hyperprolactinemia and elevated FSH levels can aid in early diagnosis and targeted management strategies to improve sexual health outcomes.

REFERENCES

1. NIH Consensus Conference. Impotence. NIH Consensus Development Panel on Impotence. JAMA. 1993; 270:83–90. [PubMed] [Google Scholar]
2. Melman A, Gingell JC. The epidemiology and pathophysiology of erectile dysfunction. J Urol. 1999; 161:5–11. [PubMed] [Google Scholar]
3. Feldman HA, Goldstein I, Hatzichristou DG, Krane RJ, McKinlay JB. Impotence and its medical and psychosocial correlates: results of the Massachusetts Male Aging Study. J Urol. 1994; 151:54–61. [PubMed] [Google Scholar]
4. Althof SE, Corty EW, Levine SB, Levine F, Burnett AL, McVary K, et al. EDITS: development of questionnaires for evaluating satisfaction with treatments for erectile dysfunction. Urology. 1999; 53:793–799. [PubMed] [Google Scholar]
5. Tellaloğlu S, Kadioğlu A. Sexual dysfunction in men, In Series of Andrology. Istanbul: Nobel Medical Co; 2000. pp. 143–147. [Google Scholar]
6. McClure RD, Marshall G. Endocrinologic sexual dysfunction. In: Singer C, Weiner WJ, editors. Sexual Dysfunction: A Neuro-Medical Approach. Armonk, NY: Futura Publishing Co; 1994. pp. 245–273. [Google Scholar]
7. McClure RD. Endocrine evaluation and therapy of erectile dysfunction. UrolClin North Am. 1988; 15:53–64. [PubMed] [Google Scholar]
8. Nickel JC, Morales A, Condra M, et al. Endocrine dysfunction in impotence: incidence, significance and cost-effective screening. J Urol. 1984; 132:40–43.
9. Foster RS, Mulcahy JJ, Callaghan JT, et al. Role of serum prolactin determination in evaluation of impotent patient. Urology. 1990; 36:499–501.
10. Govier FE, McClure RD, Kramer-Levien D. Endocrine screening for sexual dysfunction using free testosterone determinations. J Urol. 1996; 156:405–408. [PubMed] [Google Scholar]
11. Rosen RC, Riley A, Wagner G, Osterloh IH, Kirkpatrick J, Mishra A. The International Index of Erectile Function (IIEF): a multidimensional scale for assessment of erectile dysfunction. Urology. 1997; 49:822–830. [PubMed] [Google Scholar]
12. Cappelleri JC, Siegel RL, Glasser DB, Osterloh IH, Rosen RC. Relationship between patient self-assessment of erectile dysfunction and the sexual health inventory for men. Clin. Ther. 2001; 23:1707–1719. [PubMed] [Google Scholar]
13. De La Torre B, Sjöberg B, Hedman M, Bártfai G, Diczfalusy E. A study of the short-time variation and interrelationship of plasma hormone levels reflecting pituitary, adrenocortical and testicular function in fertile men. Int J Androl. 1981; 4:532–545. [PubMed] [Google Scholar]
14. Douglas C Montgomery, Elizabeth A Peck and Geoffrey Vining G. Introduction to Linear Regression Analysis. A John Wiley & Sons, Inc., Publication, 2006.