"TO STUDY CLINICAL FACTORS AND SERUM TESTOSTERONE IN RELATION TO ERECTILE DYSFUNCTION IN HIV INFECTED MEN AT KLE'S DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI. A ONE YEAR OBSERVATIONAL CROSS- SECTIONAL STUDY."

By

REG. NO.BG0118008

Dissertation

Submitted to the
KLE Academy of Higher Education and Research,
Belagavi, Karnataka.

In partial fulfillment
of the requirements for the degree of

M.D.
in
GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELAGAVI, KARNATAKA.

APRIL - 2021

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH, BELAGAVI, KARNATAKA

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LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
ACTH	Adrenocorticotropic hormone
ADAM	Androgen deficiency in aging male
BPH	Benign prostrate hyperplasia
ED	Erectile dysfunction
EMAS	European male ageing study
GH	Growth hormone
HAART	Highly active antiretroviral therapy
HIV	Human immunodeficiency virus
IIEF	International index of erectile function
IQR	Interquartile range
LH	Luteinizing hormone
LUTS	Lower urinary tract symptoms
MLCK	Myosin light chain kinase
MMAS	Massachusetts male ageing study
NANC	Non-adrenergic noncholinergic
NO	Nitric oxide
NRTI	Nucleoside reverse transcriptase inhibitors
PEDT	Premature ejaculation diagnostic tool
Serum Testosterone/Ft	Free Testosterone
SHBG	Sexual hormone-binding globulin
SHIM	Sexual health inventory for men
TNF	Tumour necrosis factor
TRT	Testosterone replacement therapy
TSH	Thyroid-stimulating hormone
TT	Total testosterone

ABSTRACT

Background: The role of testosterone in erectile dysfunction (ED) among the general population has been established, but among HIV, positive men are lacking. Hence, the present study aimed to study and correlate serum Testosterone hormone to ED and associated risk factors in HIV men.

Material and methods: This study was a cross-sectional study design involving 75 HIV positive patients. The study subjects were analyzed for serum Testosterone levels. Erectile dysfunction was evaluated by using the international index of erectile function (IIEF-5). P value < 0.05 was considered statistically significant. IBM SPSS version 25 was used for statistical analysis

Results: The prevalence of erectile dysfunction in the present study was 96%. The majority (54.67%) of them had mild to moderate ED, followed by 36% with mild ED, and 4% of them had no ED. Hypertension was found in 14.67% of the study population. The mean IIEF Scores to be 16.15 ± 2.93 (95% CI 15.47 to 16.82). The relation between depression severity and IIEF score was statistically significant [P value <0.001].In our study, we got a weak positive correlation between CD 4+ count and Erectile disfunctionwhen serum testosterone was normal in the age group 20-49yrs(rs Value: 0.316, P value: 0.163). There was a moderate positive correlation between Serum testosterone and EDin the study population (rs Value: 0.680, P value: <0.001).In our study, there was no correlation between age, and abnormal testosterone and ED **[**rs Value: -0.459, P value: 0.003]

Conclusion: Prevalence of ED among HIV positive patients in the current study was 96%. Serum Testosterone and depression correlated with ED and were the factors associated with ED. In our study, we got a negative correlation between CD4 +count, age and ART with ED in HIV positive patients.

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INTRODUCTION

The global prevalence of HIV/AIDS in 2019 is nearly 38 million population, were 36.2 million are adults, and 1.8 million are children less than 15 years of age.[1] India is the third largest HIV epidemic in the world, possessing 2.1million HIV patients.[2]HIV prevalence is higher among men than women, with 0.25% of men and 0.19% of women living with HIV as of 2017. This is due to high prevalence among key populations, including men who have sex with men (MSM), migrant workers and men who use drugs.[3] The quality of life, emotional well -being and physical health of these patients is compromised. So is the sexual health, especially in HIV men.[4] Sexual functioning and quality of life are interwoven phenomena.

In Human Immunodeficiency Virus (HIV) infection, sexual health has always been an issue of interest. The prevalence of sexual dysfunction has been diverse and increasing from 30% to 74%.[5,6] There is now general agreement that sexual dysfunction is more present in HIV-infected men than in non-HIV-infected men.[7] Erectile dysfunction is strongly associated with [lower urinary tract symptoms] LUTS in and the person with benign prostrate hyperplasia (BPH). In various literature, it has been found that Erectile dysfunction is common in ageing men with risk factors such as hypertension, cardiovascular disorders, cigarette smoking, obesity, dyslipidaemia, diabetes mellitus, metabolic syndrome, stress, anxiety and depression.[8] In general, sexual dysfunction in young men can be could be due to psychogenic, hormonal, medications, drug/ alcohol abuse or systemic conditions.

We have four important factors which are associated with sexual dysfunction in HIV/AIDS patients-: mental, hormonal, pharmacological, and other morbid conditions. Many HIV patients have a guilt feeling that they acquired HIV from the sexual transmission; it may be a mental factor that negatively influences their sexual

response. This type of psychological factor may explain higher rates of sexual dysfunctions in homosexual and bisexual men.[9] Earlier Hypogonadism was one of the most common causes of sexual dysfunction before the introduction of HAART. The prevalence of hypogonadism has been lowered with the introduction of HAART, but it still remains the most common endocrine disorder of HIV-infected men.[10] Moreover, estradiol is often higher in men (50% of them) on HAART, possibly due to augmented peripheral conversion of androgens to estrogens in lipid tissues.[11,12] HIV patients on HAART lipodystrophy is very common, and due to increase number of fibroblast and macrophages in lipotropic areas, testosterone gets converted to estrogen by intracellular aromatization. The pathophysiology of the sexual dysfunctions in this population is due to an increase in the level of estrogen, lipodystrophy and low sexual desire.[12]

Hypogonadism in HIV-infected men have similar features like of androgen deficiency in the general population and is characterized by loss of facial and body hair, decreased muscle mass and strength, a high percentage of body fat, low libido, erectile dysfunction, testicular atrophy, infertility and gynecomastia.[13] Depression, fatigue, low energy, poor concentration, anaemia and low bone mineral density with osteoporotic fractures.[13]

The need for the study

The prevalence of HIV in India is high, and the sexual health among this population is often less studied due to social stigma. Although there are reports of the prevalence of sexual dysfunction among HIV patients, there exist variations due to sample size across studies. There is a wide spectrum of endocrine abnormalities witnessed in human immunodeficiency virus (HIV) patients. The etiological factors such as mental status and hormonal factors in young HIV patients have shown an

important role in sexual dysfunction. The role of testosterone in erectile dysfunction have been extensively studied in the general population, while in HIV, the causes of serum decline of testosterone are still debated. Hence in the present study, we attempted to correlate erectile dysfunction with clinical factors and serum testosterone in HIV positive patients.

AIMS AND OBJECTIVES

- To correlate between erectile dysfunction with clinical factors and serum testosterone.

REVIEW OF LITERATURE

1. ERECTILE DYSFUNCTION:

a) Definition

Erectile dysfunction (ED) is defined "as the persistent inability to achieve or maintain an erection adequate for satisfactory sexual activity."[14]

b) Classification

Table 1: Classification and common causes of erectile dysfunction.[15]

Category of Erectile dysfunction	Common disorders	Pathophysiology
Neurogenic	Stroke -Alzheimer's disease - Radical pelvic surgeries - Spinal cord injury -Diabetic neuropathy -Pelvic injury	Interrupted neuronal innervation -Failure to initiate NO release
Psychogenic	-Depression -Psychological stress -Performance anxiety -Relationship problems	Impaired nitric oxide (NO) release -Loss of libido -Sympathetic nervous system activation
Hormonal	Androgen Deficiency -Hyperprolactinemia -Diabetes -Chronic opioid use	Loss of libido -Inadequate NO release -Morphological changes in the penis (atrophy)
Vasculogenic (arterial and cavernosal)	-Hypertension -Atherosclerosis -Hyperlipidemia -Diabetes mellitus -Obesity -Trauma/pelvic fracture -Tobacco Use -Peyronie's disease	Impaired penile veno-occlusion -Inadequate arterial inflow
Drug-induced	Antihypertensives -Antiandrogens -Antidepressants -Alcohol abuse	Central Nervous System suppression -Decreased libido -Alcoholic neuropathy -Vascular insufficiency
Systemic diseases	Ageing -Diabetes mellitus -Chronic renal failure -Generalized atherosclerotic disease	Multifactorial -Neuronal and vascular dysfunction

c) **Epidemiology**

Numerous studies have been researching on the epidemiology of erectile dysfunctions in different ethnics and conditions. A known fact is that erectile dysfunction is more prevalent in men of older age given by: the European Male Ageing Study (EMAS) and the Massachusetts Male Ageing Study (MMAS).[16] The MMAS exhibited an overall frequency of mild to moderate ED of 52% in men aged 40–70 years; and suggested that ED was significantly associated with overall health, age and mental health.[17] In contrast, the EMAS, the major European multicentre population-based study of elderly men (40–79 years) which is based on various age subcategories, stated that a frequency of ED ranging from 6% to 64% is increases with age and with an average prevalence of 30%.[16]

There are limited studies on the prevalence of ED globally.[18,19,20] From these limited studies it can analyze that there is a greater prevalence of ED in the United States and eastern, Southeast Asian compared to Europe and South American countries. This could be attributed to variations in the socioeconomic status, ethnicity. Thus, it is recommended to research and explore other factors such as environmental and genetic influence on ED.

Reports from USA, Netherlands and Brazil found the incident cases ranging from 19 to 66 per 1,000 men each year.[21,22] however, these studies have shown the lesser duration of follow up with non- uniformity in age and with restricted places of subjects. The evidence from epidemiology have showed a significant association between ED and lower urinary tract infection (LUTS)along with benign prostatic hyperplasia (BPH) and LUTS related to obstruction of urine produced by BPH. This suggestion was upheld even after correcting for probable confounding factors, such as comorbid and age.[18,23]

Erectile dysfunction and LUTS found in BPH commonly are encountered in aging and aged men where other factors at risk such as diabetes mellitus, cardiovascular disorders, smoking, lipid dysfunction, stress, metabolic syndrome, depression, obesity and hypertension.[8]

Erectile dysfunction studied systematically in men greater than 40 years of age[16], the occurrence of ED in young adults is infrequently viewed to be of interest.[24, 25] From a realistic experience of the researcher through his study found that one in four men consulted him for ED and were aged less than 40 years. [26] From another study, it was found that 22.1% of men less than 40 years had low (less than 21) Sexual Health Inventory for Men (SHIM) scores.[27] All the data which is captured were based on the real-life experience of the practitioners and were not well appreciated by large cohort studies and prospective studies, thus more young men with ED were related to psychological reasons.The hints to the basis of psychogenic cause can be included with good quality impulsive, self-stimulated and sudden onset, chief life situations or past mental illness. Other cause which includes anxiety, lack of emotional awareness in young adults with ED can be a symptom of a underlying serious organic conditions.[28,29]

d) **Risk factors**

The documentation of the exact risk factors and pathogenetic reasons involved in ED is the basis of exact diagnosis and effective treatment.

Lifestyle factors:

Alcohol, Diabetes mellitus and smoking habits have steadily been exposed to its effect on the erectile role. Direct association of dose-dependent on smoking and alcohol was found to effect ED.[30] Few studies have also supported the result of alcohol use.[31,32] Intake of low protein, low fibre diet and high on trans-fat and refined sugar intake were also the related risk of developing ED.[31] From a meta-analysis, it was observed that good physical activity decreases the risk of developing ED.[33]The cross-sectional and prospective studies have shown an association of metabolic syndrome and obesity with ED.[34]It is well known that obesity-related to hypogonadism and greater risk of cardiovascular disease, associated with greater frequency of ED in men who were obese. But from the latest clinical studies showed that association between ED and central obesity was independent of obesity-related hypogonadism and comorbidities. [34] A hypothesis of increasing levels of TNF (tumour necrosis factor- cytokine involved in inflammation) may be a moderator for these settings. [35] but however future studies can only confirm this. Type 1 and 2 Diabetes mellitus, along with pre-diabetic, can also be a factor to influence sexual life.[36] Other diabetic-related sexual impairment include atherosclerosis, neuropathy, endothelial dysfunction and hypogonadism.[36,34,37]

Cardiovascular disease:

Arteriogenic erectile dysfunction and cardiovascular disease are measured diverse manifestations of a common underlying vascular disorder. From a 3 independent meta-analysis, it is suggested, that ED to be considered as an indicator for (coronary heart disease)[38,39], and as a predictor of upcoming asymptomatic cardiac events.[40]This suggestion is predominantly important in younger men aged less

than 55 years and those with EDand no other comorbidities[41], highlighting the role of early diagnosis and meticulous management of ED-associated morbidities. Consequently, the Princeton III Consensus Recommendations for the managing ED and cardiovascular disease specify that incident ED has an alike, or even better, predictive value for CVD than other traditional risk factors such as smoking, hypertension, diabetes or dyslipidaemia.[42]

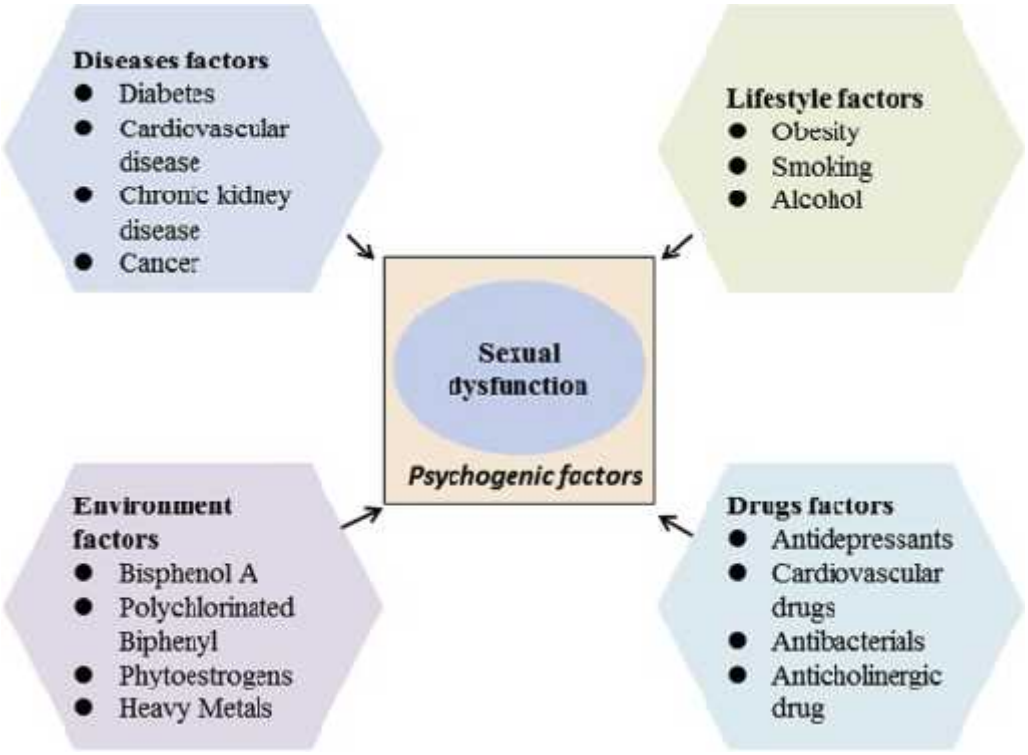
BPH and LUTS:

As mentioned previously, lower urinary tract infection in BPH is well associated with ED. It is well noted that LUTS can be used as an independent risk factor for developing ED in men.[18]. However, its pathophysiology is not known.

Psychogenic and relationship factors:

The psychogenic and rapport spheres need to be assessed in men with symptoms of ED. The most organic cause, such as diabetes for ED, can also contribute to stress and psychological imbalance. Anxiety to perform sex is another frequently encountered reason for ED leading to low self- esteem, depression and abstain from sex. Several psychotropic drugs promote sexual disturbances and erectile disturbances.[43]

Figure 1: Factors that determine sexual dysfunction.[44]

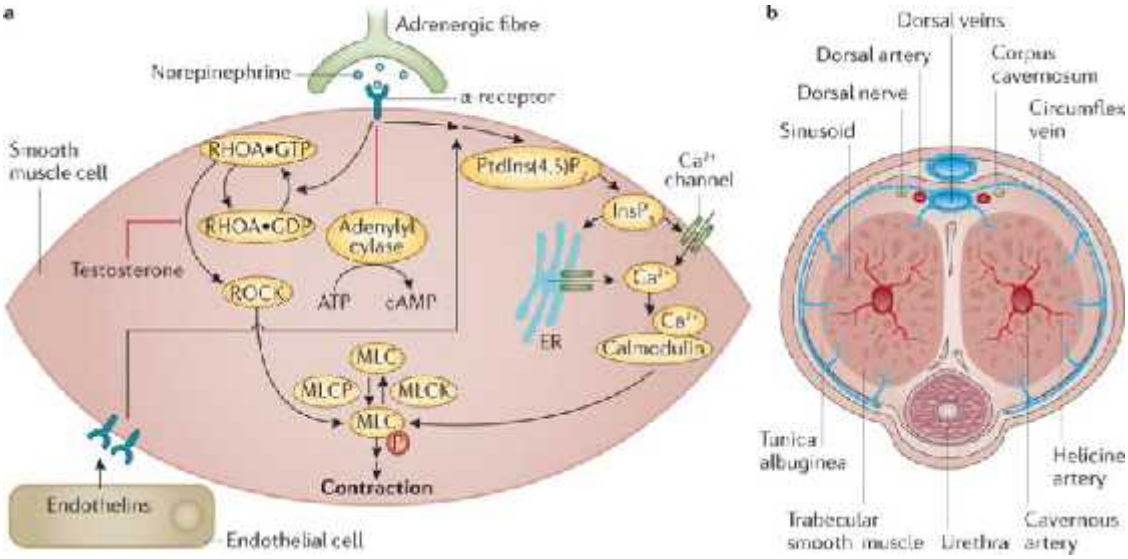


e) <u>Pathophysiology</u>

The penis remains in the flabby state when the smooth muscle is contracted. The smooth muscle contraction is controlled by an amalgamation of adrenergic (noradrenaline) control, endothelium-derived contracting factors (prostaglandin and endothelins and intrinsic myogenic control.[28] during the sexual drive the erection happens after the release of nitric oxide (NO) from the non-adrenergic non-cholinergic (NANC) nerve fibres. Later acetylcholine is released from parasympathetic cholinergic nerve fibreswhich result in the activation of signaling pathways with decreased intracellular Ca^{2+} levels and increased cyclic GMP (cGMP) concentrations causing smooth muscle cell easing.[45] After the relaxation of smooth muscle blood starts to fill in the lacunar spaces of corpora cavernosa, that cause a compression of the sub-tunical venules, thus hindering the venous outflow (veno-

occlusion). This cycle is reversed as cGMP is hydrolysed by phosphodiesterase type 5 (PDE5).[45]ED can result when either of these events is disturbed.

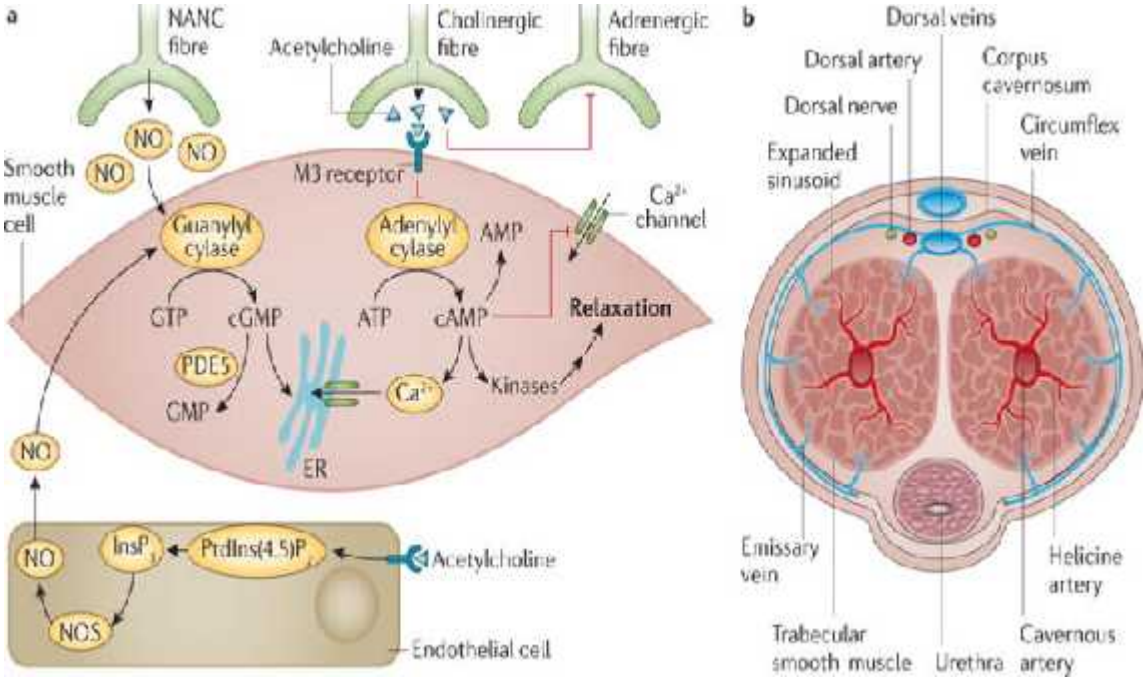
Figure 2: A diagrammatic explanation of penile smooth muscle contraction -: the flaccid state:



|**"a.** Ca^{2+} influx into cells is regulated by noradrenaline signalling and levels of inositol-1,4,5-trisphosphate ($Ins(1,4,5)P_3$, which is produced from phosphatidylinositol-4,5-bisphosphate ($PtdIns(4,5)P_2$) by phospholipase C) in the cells; increased intracellular Ca^{2+} binds to calmodulin, facilitating the formation of the calmodulin–myosin light chain kinase (MLCK) complex. This leads to the phosphorylation of MLC, resulting in smooth muscle contraction and a flaccid penis. Noradrenaline signalling also inhibits adenylyl cyclase and modulates the RHO-associated protein kinase (ROCK) pathway, which increases the sensitivity of MLC to Ca^{2+}, a process negatively regulated by testosterone. Endothelins and prostaglandins from the endothelium also trigger an increase in intracellular Ca^{2+} to promote smooth muscle contraction. **B** | When the smooth muscle is contracted, the inflow of blood through the cavernous artery is minimal, and blood outflows freely through the sub-

tunical venular plexus. ER, endoplasmic reticulum; MLCP, myosin light chain phosphatase".[46]

Figure 3: Penile smooth muscle relaxation -: the erect state



"a | Upon sexual stimulation, a normal erection occurs after nitric oxide (NO) release from non-adrenergic non-cholinergic (NANC) nerve fibres causes the activation of guanylyl cyclase, which raises the concentration of cyclic GMP, and after parasympathetic cholinergic nerve fibres release acetylcholine, which activates adenylyl cyclase to increase the levels of cyclic AMP. Signalling pathways that are triggered decrease intracellular Ca^{2+} levels and lead to smooth muscle cell relaxation. **B** | As the smooth muscle relaxes, blood is able to fill the lacunar spaces in the cavernosa, leading to compression of the subtunical venules, thereby blocking the venous outflow. The process is reversed as cGMP is hydrolysed by phosphodiesterase type 5 (PDE5). ER, endoplasmic reticulum; $InsP_3$, inositol trisphosphate; NOS, NO synthase; PtdIns(4,5)P_2, phosphatidylinositol 4,5 bisphosphate".[46]

2. ERECTILE DYSFUNCTION IN HIV:

2a) Epidemiology, Pathophysiology:

From the previous studies, it is evidenced that highly active antiretroviral therapy (HAART) to ensure the best care to HIV men as this decreases the prevalence of hypogonadism and controlling the infection.[47] However, the frequency of sexual dysfunctions in recent years with HAART have shown varied frequencies due to inconsistencies in the methodology of studies, ethnic. These studies have shown the prevalence of ED ranged between 9-74%, low sexual desire 24-73% and ejaculatory problems in 36-42%.[47]A Clinical study done in 2001 on 78 HIV gay men found 69% of men to have one and more sexual disturbances. The prevalence of ED was 38% and slowly raised to 51% due to the use of condoms. Less interest in sex was found in 41% and delayed ejaculation in 24%.[48] Another,cross-sectional study on the Brazilian population with AIDS showed that nearly 50% of the men informed ejaculatory symptoms, 33% stated ED, and 12% stated dyspareunia.[49]

It is well noticed that individuals diagnosed with HIV infection by itself negatively effects the psychology of these patients leading reduced sexual urge and those who are active have unprotected sex.[6] practice of safe sex post-HIV diagnosis has also added to negate the sexual role in the majority of individuals.[6]However, subjects who have partners remain active in sexual life than those who do not have.[50]

2b) Risk factors:

The 4 important factors related to sexual impairment in HIV men are: mental, hormonal, pharmacological, and other morbid conditions.

Mental Factors:

A feeling of discrimination due to HIV infection and side effects of lipodystrophy prior to 12 months of the survey have shown low sexual performance

even though they had partners.[51]A guilt feeling of having acquired infected through sexual contact lowers sexual activity and leads to mental stress. This is well documented in both bisexual and homosexual men.[9]Depression and anxiety both contribute to less sexual activity.[52] Depression, older age and greater CD4 count was found not to be related to ED, but, found high CD4 count to guard ED.[53]

Hormonal Factors:

Before the introduction of HAART, hypogonadism was the most common cause of sexual impairment in HIV individuals. However, in recent times, high testosterone levels may be noted in infected HIV individuals than non -infected subjects. The introduction of HAART has decreased the prevalence of hypogonadism, but yet endocrine problems persist in HIV subjects.[10] individuals on HAART have higher levels estradiol due to increased conversion androgens to estrogens in the lipid tissues.[11,12]High prolactin is associated with sexual impairment as it lowers the release of GH gonadotropins.[6]

Pharmacological Factors:

Drugs suchas fluconazole, ketoconazole, ganciclovir, methadone, megestrol and cimetidine may lower the testosterone levels and cause sexual dysfunction.[6] Diuretics, Antihypertensives, benzodiazepines, hypolipemic, antipsychotics antidepressants are also linked with sexual impairment.[5]

Comorbid Conditions:

Morbid conditions are common in HIV-infected individuals, and some of them are often associated with sexual dysfunction, those morbid conditions are hepatopathy, diabetes mellitus, hyperlipidemia, hypertension, cardiovascular disease, and alcohol dependence.[54] Peripheral neuropathy is a well-documented complication of both HIV infection and HAART, most common with thymidine analogue

nucleoside reverse transcriptase inhibitors(NRTI). There is a significant association between peripheral neuropathy and delayed ejaculation in HIV men.[55] Another study on 12 patients with ED reported neuropathy of the sacral region was associated with protease inhibitor use, particularly indinavir.[56]

Pathophysiological Issues:

Significant autonomic dysfunctions were seen in HIV-infected individual.[6] This result may describe the relation between delayed ejaculation and peripheral neuropathy.[55]The physiological process of ejaculation is under autonomic control through the hypogastric (sympathetic) and pudendal (parasympathetic) nerves.[55] Another related point concerning the pathophysiology of the sexual impairment in these individuals is that several subjects on HAART to treat their HIV infection have lipodystrophy and have high estrogen levels and report low sexual desire. An explanation to this is given by a mechanism: with increased activity and number of macrophages and fibroblasts in the lipodystrophy areas where the majority of the testosterone is converted to estrogen by an aromatization mechanism intracellularly. This process is further augmented by tumor necrosis factor interleukin 6, and hydroxy corticosteroid is already there in HIV patients with lipodystrophy.[12]In older individuals it was found that testosterone was decreased and estradiol raised in relation to ED.[57] Circumstantial evidence propose a correlation between protease inhibitor use and sexual impairment. However, this finding is supported by only a few studies.[58,6]

Clinical presentation and diagnosis of Hypogonadism in HIV:

Hypogonadism was reported high in HIV men prior to the introduction of HAART treatment especially in men whose immune suppression was in advanced stages ($CD4 < 100\ cells/mm^3$).[59] in the past the estimated hypogonadism occurred in

30 to 50 % of AIDS-related wasting.[60] Ever since the HAART therapy is given at the starting stages of HIV compared to past the prevalence of hypogonadism is lessened with only 20% having it who have received HAART.[61]

HIV individuals with hypogonadism present with loss of body and facial hair, with low muscle mass and strength, increased body fat, decreased libido, ED, infertility and rarely gynecomastia.[13] Fatigue, Depression, poor concentration, low energy, low bone mineral density with osteoporotic fractures and mild anemia.[13]

There is no specific guidelines or protocols in the diagnosis of hypogonadism. But a cut-off for general population is frequently suggested who are suspicious for hypogonadism clinically: that is later should be confirmed with a low serum total testosterone level (generally <300 ng/mL) in two different samples in the morning (8:00 h).[62] There is increased sexual hormone-binding globulin (SHBG)among HIV patients and therefore a free testosterone levels should be checked coz the HIV patients may have normal total testosterone levels, but free testosterone levels are low.[63]

Low levels of testosterone along with low or normal LH and FSH (gonadotropins) are indicative of hypogonadotropic hypogonadism. These patients should be further analysed for some reversible causes of deficiency in gonadotropin, for example, hyperprolactinemia.[63] Cortisol, growth hormone (GH), Adrenocorticotropic hormone (ACTH), thyroid-stimulating hormone (TSH) and free T4 (FT_4), insulin growth factor-1 (IGF-1), should be further investigated.[60] other investigation like MRI of the pituitary is recommended in subjects with a diagnosis of hypogonadotropic hypogonadism to exclude hypothalamic or pituitary lesion.[60]

Medication management of hypogonadal status in HIV subjects has shown to improve muscle strength and mass and QOL and indices of depression and bone mass.[63]

Subjects with other hypogonadal symptoms, low libido, low body mass or low weight and loss of bone mineral density in spite of viral suppression on HAART, are suitable for testosterone replacement therapy. [63] Though it is usually well endured, testosterone-related side effects include oiliness of skin, acne and on long usage leads to testicular atrophy and erythrocytosis.[63]

CORRELATION BETWEEN ERECTILE DYSFUNCTION AND SERUM TESTOSTERONE IN HIV:

Testosterone – structure, function in short:

Testosterone is a primary male hormone indicative of sex differentiation and is capable of constructing male features, fertility and spermatogenesis. The effects of Testosterone are evident in the initial development of fetus that is in the first six weeks of life where the reproductive tissues of male and female are alike. And at the seventh week of life in utero, the SRY (sex-related gene on the Y chromosome) is responsible for the growth of the testicles. Seminiferous tubules are developed from the Sertoli cells of the fetal testicles. The substance released by Sertoli cells called the "Mullerian inhibiting substance" helps to deteriorate female structures such as the uterus, fallopian tubules and vagina. The endothelial cells and fetal Leydig cells travel to the gonad and start making testosterone to support the differentiation of the Wolffian duct (paramesonephric duct) structures that become distinctive to the male urogenital tract. Further, this testosterone converted to DTH encourages to form prostrate and external male genitalia. Its function goes on to help in descending the

inguinal canal during the last trimester of pregnancy. A lack of Y chromosome is deficient of the SRY gene and thus develop ovaries, and these ovaries produce insufficient testosterone where wolffian ducts are not produced. Thus, producing female reproductive structures.[64]

Role of Testosterone:

Androgens have always been assumed to play a major role in male erectile function because:

- There is a decrease in serum testosterone levels with aging and a time period when the prevalence of ED increases.
- Castration usually causes a decline in sexual function.
- The sexual function returns to normal in castrated (severely hypogonadal) men who undergo treatment with exogenous androgens.[65]

.**Why there is a premature decline of testosterone in HIV:**

In the majority of HIV patients with low testosterone level have unexplainable low luteinizing hormone (LH), where an aberration of pituitary gonadotrophin secretion can be explained or hypothesized. Hence the hypothalamic-pituitary axis should be watched as the key elements involved in the development of T deficiency in HIV-infected patients.[66] BMI and visceral fat are found to forsee and contra wisely related to total serum testosterone in HIV men. Suppression of the hypothalamic axis also suggests decreasing testosterone hormone.[67]

In the absence of obvious obesity, fat relocation due to HIV-related lipodystrophy leads, to increased visceral adiposity. Estradiol is identified to exert a strong repressive effect on LH secretion by acting on both hypothalamus and the pituitary in men[66], the augmented serum estradiol levels related to adiposity could

describe secondary hypogonadism in HIV men.[67] High visceral adiposity and increased Testosterone aromatization into estradiol, contribute to increased serum estradiol levels in HIV men with secondary hypogonadism.so testosterone deficiency and visceral adiposity can be reversed through weight loss and visceral fat reduction.[66]

MOST RELEVANT STUDIES:

GLOBAL:

A cross-sectional study by **Gomez T et al[68] 2019,** aimed to find the occurrence of ED and risk factors associated among HIV positive men in Brazil. A total number of participants recruited for the study were 134, and the mean age was 44.8 years. The prevalence of ED among the population was 21.6%, and among then 86% had severe ED were the majority of them belong to low socioeconomic status with low educational status. However, this study found no correlation with ED and morbid conditions, age and ART therapy. Hence this study results found low economic status and unemployment to be the main factors related to ED in HIV positive men than other health-related factors and antiretroviral therapy.

A cross-sectional observational study by **Fumaz, C et al[4] 2017,** studied the prevalence and clinical factors of ED among HIV patients. this study involved 501 subjects with a median age of 42years (IQR 35,48). The mean duration of diagnosis of HIV was 6.3 years. This study found 58.5% of ED among the study population, with 30.1% with mild, 19.5% with mild to moderate, 6.1% with moderate and 2.5% with severe ED. Nearly 19% of the study population were on ED medications. From the analysis, it was found that all the variables correlated well with all ED classification were of the old aged group, with increased time of diagnosis of HIV, nonadherent to

ART, greater scores of HAD, infection with Hepatitis C infection and lower CD4 counts. From the multivariate analysis, it was found that all classification of ED except mild ED correlated well with old age and greater scores of HAD. Hence this study concluded that ED was more affected in well-controlled HIV patients with psychological factors playing a key role in ED among HIV patients.

A study by **Gomes et al**[69] **2016,** accessed the occurrence of testosterone deficiency and risk factors among HIV men. This study included 245 men with HIV infection. The prevalence of low testosterone was found in 29.4% were 56.9% were found to have hypogonadotropic dysfunction, and 43.1 % with hypergonadotropic dysfunction. Subjects with testosterone deficiency were old aged and had high levels of HbA1c and hypertension ($p < 0.001$). Visceral fat accumulation was prevalent in patients with low testosterone levels and in addition, poised greater risk for cardiovascular disease. Hence the deficiency of testosterone hormone in HIV patients can lead to hypogonadisms in the majority of HIV men.

A cross-sectional study by **Monroe, A et al**[70] **2014,**aimed to compare and find the occurrence of hypogonadism in HIV men with non -infected HIV men. This study also analysed free (FT) and total testosterone (TT). A total of 530 men underwent Testosterone measurements with the sample given during morning, and 68.7% of them were HIV positive. The frequency of biochemical hypogonadism was in HIV-infected was 9.3%, and HIV-uninfected was 7.2%. However, the frequency of hypogonadism was high (24%) in men with testosterone replacement therapy (TRT) compared to HIV negative men (7.8%). This study found the frequency of biochemical hypogonadism in HIV positive cases was 10% with normal levels of TT with low FT.

A systematic review by **Ashby J et al**[71] **2014,** assessed the androgens in HIV men. This review found a higher prevalence of androgen declines in HIV men compared to non- infected population. The deficit androgen correlated to well with low levels of luteinizing hormone and FSH follicle-stimulating hormone. Etiology for the decline in Testosterone levels was found to be chronic diseases, increased viral load, ART therapy, secondary infections, comorbid conditions with coexisting infection and age-related.

A cross-sectional observational study by **PérezI et al**[72] **2013,** accessed the incidence of ED and factors associated with ED in HIV men with no symptoms. A total of 158 HIV positive men were included in the analysis. The mean age of the study population was 46.0 years were 96.2% were taking antiretroviral therapy, and the mean CD4 count was 534 cells/mL. The prevalence of ED was 67.1%. The factors associated with ED were age (OR 4.5 for each 5 years; 95% CI 4.3-4.7; p=0.0001) and anxiety (OR 8.2, 95% CI 2.2-30.4; p=0.002). Hence this study results suggest that HIV men had a high prevalence of ED even with good control of the viral load. Anxiety and ageing factors were the contributing factors for ED in HIV men.

A cross-sectional study by **Zona S, et al**[7] **2012,** aimed to evaluate and compare the prevalence of ED among young to middle age HIV men with that of the control group(without HIV infection) utilizing IIEF 15 questionnaire. This study involved 444 men with HIV infection and 71 men without HIV infection. The results of this study found the control group to be much younger than HIV infected men and displayed greater BMI. However, the frequency of different grades (mild, moderate and severe) was greater in HIV infected men at all age groups. From the multivariable analysis, HIV infection was found to be a strong predictor of. ED. Hence this study results suggest that HIV infection by itself is a strong predictor for ED.

A systematic review by **Scanavino M et al**[6] **2011,**aimed to analyze the role and correlation of HAART with sexual dysfunction in HIV men.This review showed deficiencies in the standardizing measure, study design which resulted in contradictory results.

A cross-sectional study by **Rochira V et al**[73] **2011,** aimed to evaluate the gonadal status of HIV men and access the Testosterone levels with associated factors for low Testosterone levels. This study involved 1325 patients with the majority of them having lipid imbalance. At the same time, 247 patients were assessed for their sexual behavior. Low Testosterone levels were observed in 212 patients, in the age group of 40-59years of age. Low Testosterone levels had correlated with low and normal LH. Increased fat accumulation was associated with low Testosterone levels of individuals. However, osteoporosis was significant in both the group of patients with Testosterone deficiency. Hence this study results found a premature decline in Testosterone levels in young individuals infected with HIV and correlated with visceral fat.

INDIANstudies:

A cross-sectional study by**Pongener N et al**[74] **2019,**aimed to find the frequency of hypogonadism in HIV subjects. This study recruited HIV positive males aged 18–65 years who were on ART. Androgen Deficiency in Ageing Male (ADAM) questionnaire tool was used to analyse hypogonadism. Among 426 participants, 120 subjects who had possible hypogonadism were analysed. The prevalence of hypogonadism was 23.3% were 85.7% found to have secondary hypogonadism. A significant correlation was found between CD4 count and hypogonadism, with correlation with BMI and ART treatment duration.

A study by **Dutta D et al**[75] **2017,** aimed to evaluate the occurrence, pattern and predictors of hypogonadism in HIV-infected Indians. This study analyzed 359 subjects (225 males; 134 females). The mean duration of diseases was 61.44±39.42 months, and 88.58 % were on highly active antiretroviral therapy (HAART), TB was present in 40.67 %, and VIT D deficiency was present in 89.69 %. Low Testosterone levels were found in 39.11% of the male population. The prevalence of primary hypogonadism was 7.56%, hypo trophic hypogonadism was 31.56%, and compensated hypogonadism was 12.44%. This study showed older males to be affected by hypogonadism with high opportunistic infections with long duration of disease.

A study by **Pathak A et al**[13] **2015,** aimed to assess the gonadal function with HAART therapy in HIV males. The total number of subjects involved in this analysis were 45 and were grouped based on CD4 count. The group A consist of patients with CD4 count ≤200 and group B consist of patients with >200 cells/μL. The Testosterone levels at baseline before initiation of ART was measured. Group A consisted of 29 subjects with Testosterone levels >200 ng/dL, were among these subjects, 66.67% had decreased LH and FSH levels. Group B consisted of 16 subjects with no one having decreased Testosterone levels and only 12.5% and 43.8% had decreased LH and FSH levels. The overall prevalence of hypogonadism in the study population was 13.33%. The study results showed good improvement in the Gonadal function with HAART therapy.

A prospective study by **Tripathy R et al**[76] **2015,**aimed to access the adrenal, thyroid and gonadal dysfunction and correlate with different levels of CD 4 counts in the incident cases of HIV. This study involved 43 HIV patients and was group into 3. Majority 88.3%of the study population had gonadal dysfunction with increased

frequency of thyroid dysfunction in 60.4% and adrenal insufficiency in 27.9%. The majority had secondary hypogonadism 68.4% than primary 31.6%. There was no significant difference in the hormonal imbalance between male and female ($P > 0.05$). Around 27.9% had several hormonal imbalances. This study found no correlation between serum hormonal levels and CD4 count. Hence this study results suggest the prevalence of hormonal imbalance in HIV infected cases with no association of CD4 count with hormones.

A study by **Meena LP et al**[77] **2011,** aimed to find the frequency of thyroid, gonadal and adrenal dysfunction in HIV males and further to assess the endocrine function across the various level of CD4 cell counts. This study involved 150 subjects and was grouped into group A consisting of HIV subjects with CD4 count less than 200/mm, and group B consisting of HIV subjects with CD4 count greater than 200/mm CD4 count 200-350.mm3. Group C is having HIV subjects with CD4 count>350/mm3. The group A consisted of 50 subjects where 23 of them displayed basal cortisol levels greater than 25 microg/dl. Nearly 15 of the cases had subclinical hypothyroidism, and 11 had overt hypothyroidism. 25 cases in group A had gonadal dysfunction where the majority had primary gonadal dysfunction with high LH levels. However, in group B, none of them exhibited hypercortisolism, but 11 of them had risen cortisol levels, 18 showed subclinical hypothyroidism and 17 had hypogonadism all showed a rise in LH. The group C with 50 subjects, 2 of them had hypercortisolism, 5 of them had risen cortisol, 12 showed subclinical, and one with overt hypothyroidism and 7had primary hypogonadism, and 1 had secondary hypogonadism. There was a strong reverse correlation between CD4 count and basal cortisol, LH and TSH, but the direct correlation with serum T levels. This study

results showed a high prevalence of endocrine dysfunction among HIV positive subjects

A study by **Shindel et al**[78] **2011,** assessed the relationship between HIV and sexual dysfunction (especially in MSM) using different validated tools. This survey was based on the informational data perceived through internet sources. Two validated tools used were the International Index of Erectile Function (IIEF) for MSM and the Premature Ejaculation Diagnostic Tool (PEDT) in subjects to classify the risk of sexual dysfunction. This study involved 1361 men, where 236 were HIV infected. This analysis showed a greater occurrence of ED in men with the advancement of HIV infection in 40to 59 years of age compared to non -HIV patients.

LACUNAE IN LITERATURE:

Literature till date has shown decreased testosterone in HIV patients leading to sexual dysfunction among them. However, in Indian HIV subjects, the correlation of serum testosterone and clinical features in sexual dysfunction is less studied. Hence, more studies in this regard will help in rectifying the etiology and diagnosis of sexual dysfunction and thereby improving their overall quality of life among HIV patients.

MATERIALS AND METHODS

Study site: This study was conducted in the department of General Medicine at Jawaharlal Nehru Medical CollegeKAHER University and KLE'SDR.PRABHAKAR KORE HOSPITAL AND MRC, Belgaum.

Study population: All the eligible HIV positive patient aged greater than 18 years of age attended to the department of General Medicine at Jawaharlal Nehru Medical Collegewere considered as the study population.

Study design: The current study was a hospital based observational cross- sectional study.

Sample size: 75 patients

Sample Size was calculated by the following formula:

$$n = 4 \times p \times q / d^2$$

Where p=Prevalence of HIV infected men (50 %)

q = 100 – p d = absolute error (1.e., 7)

Sampling method: All the eligible subjects were recruited into the study consecutively by convenient sampling till the sample size is reached.

Study duration: The data collection for the current study was done between January 2019 to December 2019 for a period of 1 year.

Inclusion criteria:

1. All documented HIV infected men > 18 Years

Exclusion criteria:

1. Chronic liver disease
2. Renal failure
3. Diabetes
4. Hypertension (on beta-blockers)
5. Elderly(Age >65 yrs)
6. Psychiatry disorders
7. Genital abnormalities
8. Men on Medication that could influence erectile like ketoconazole, ganciclovir, methadone, anti-depressants and beta blockers.

Ethical considerations: Study was approved by the institutional human ethics committee. Informed written consent was taken from all the study participants. Only those participants willing to sign the informed consent were included in the study. The risks and benefits involved in the study and the voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters and investigations CBC,LFT,,RFT, S.Free Testosterone, usg abdomen chest x-ray and CD4 count were documented in a study proforma. Erectile dysfunction was evaluated by using the international index of erectile function (IIEF-5)

Methodology: All the Patients fulfilling the inclusion criteria and willing to participate were included in the study. Informed consent was obtained, and Further, they were be subjected to a detailed history and predesigned proforma. Data were analyzed and tabulated for factor taken into consideration.

Statistical Analysis:

IIEF-5 score was considered as the primary outcome variable. PHQ-9, Serum Free Testosterone, CD4 Count Hemoglobin, etc., were considered as secondary outcome variables. Age, BMI were other explanatory variables.

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented by using appropriate diagrams like bar diagram and pie diagram.

All Quantitative variables were checked for normal distribution within each category of an explanatory variable by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test p value of >0.05 was considered as a normal distribution.

The association between explanatory variables and categorical outcomes was assessed by cross tabulation and comparison of percentages. Odds ratio, along with 95% CI, is presented. Chi square test was used to test statistical significance. The association between variables for non- normally distributed Quantitative parameters, Medians and Interquartile range (IQR) were compared between study groups using the Kruskall Wallis Test. Association between quantitative explanatory and outcome variables were assessed by calculating the Spearman correlation coefficient, and the data was represented in a scatter diagram.

P value < 0.05 was considered statistically significant. IBM SPSS version 25 was used for statistical analysis.[79]

RESULTS

A total of 75 subjects were included in the final analysis.

Table 2: Descriptive analysis of Age in the study population (N=75)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
Age	46.12 ± 8.49	47.00	21.00	60.00	44.17	48.07

The mean Age was 46.12 ± 8.49 years in the study population, the minimum Age was 21 years, and the maximum Age was 60 years in the study population (95% CI 44.17 to 48.07). (Table 2)

Table 3: Descriptive analysis of BMI in the study population (N=75)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
BMI	23.2 ± 2.58	23.70	18.60	28.80	22.61	23.79

The mean BMI was 23.2 ± 2.58 in the study population, minimum BMI was 18.60, and maximum BMI was 28.80 in the study population (95% CI 22.61 to 23.79). (Table 3)

Table 4: Descriptive Analysis of Gender in the study population (N=75)

Gender	Frequency	Percentages
Male	75	100.00%

Among the study population, all the participants were male. (Table 4)

Table 5: Descriptive analysis of questionnaire in the study population (N=75)

Questionnaire	Frequency	Percentages
Q1		
3	26	34.67%
4	40	53.33%
5	9	12.00%
Q2		
2	9	12.00%
3	39	52.00%
4	26	34.67%
5	1	1.33%
Q3		
2	22	29.33%
3	38	50.67%
4	15	20.00%
Q4		
2	14	18.67%
3	35	46.67%
4	24	32.00%
5	2	2.67%
Q5		
2	10	13.33%
3	45	60.00%
4	19	25.33%
5	1	1.33%

Among the study population in Q1, 26 (34.67%) gave IIEF score of 3, 40 (53.33%) gave IIEF score of 4, 9 (12%) gave IIEF score of 5. In Q2, 9 (12%) gave IIEF score of 2, 39 (52%) gave IIEF score of 3, 26 (34.67%) gave IIEF score of 4, 1 (1.33%) gave IIEF score of 5. In Q3, 22 (29.33%) gave IIEF score of 2, 38 (50.67%) gave IIEF score of 3, 15 (20%) gave IIEF score of 4. In Q4, 14 (18.67%) gave IIEF score of 2, 35 (46.67%) gave IIEF score of 3, 24 (32%) gave IIEF score of 4, 2 (2.67%) gave

IIEF score of 5. In Q5, 10 (13.33%) gave IIEF score of 2, 45 (60%) gave IIEF score of 3, 19 (25.33%) gave IIEF score of 4, 1 (1.33%) gave IIEF score of 5. (Table 5)

Table 6: Descriptive analysis of IIEF score group in the study population (N=75)

IIEF Score Group	Frequency	Percentages
Moderate ED	4	5.33%
Mild-moderate ED	41	54.67%
Mild ED	27	36.00%
No ED	3	4.00%

Among the study population, 4 (5.33%) of the participants had Moderate ED, 41(54.67%) of the participants had Mild-moderate ED and 27 (36.00%) of the participants had Mild ED. (Table 6 & Figure 4)

Figure 4: Pie chart of IIEF score group in the study population (N=75)

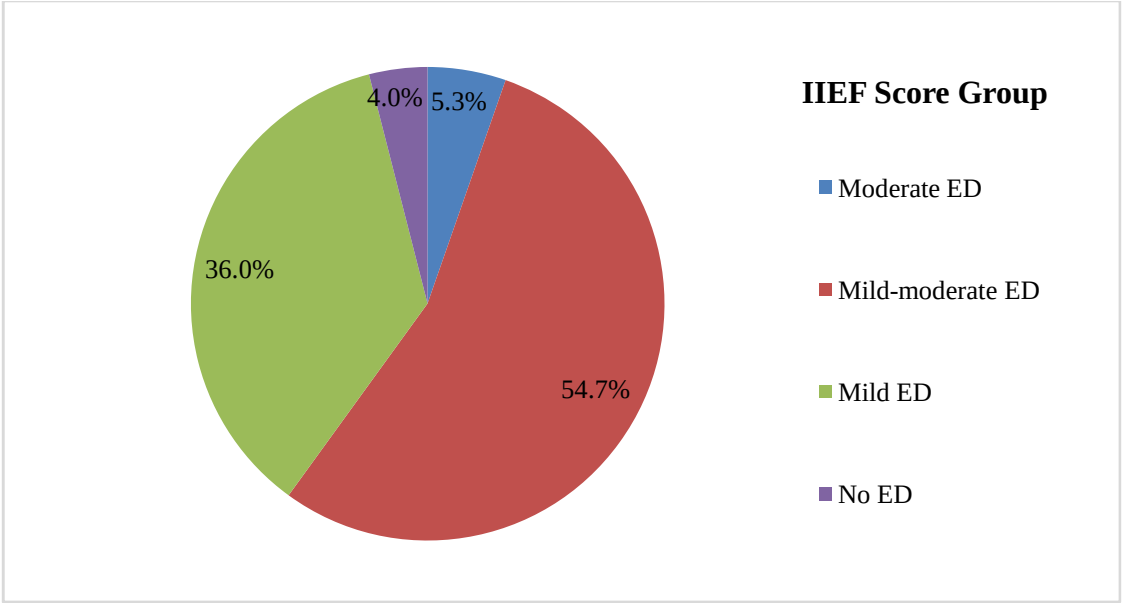


Table 7: Descriptive analysis of IIEF scores in the study population (N=75)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
IIEF Scores	16.15 ± 2.93	16.00	11.00	22.00	15.47	16.82

The mean IIEF Scores was 16.15 ± 2.93 in the study population, minimum IIEF Scores was 11, and maximum IIEF Scores was 22 (95% CI 15.47 to 16.82). (Table 7)

Table 8: Descriptive analysis of Depression severity in the study population (N=75)

Depression Severity	Frequency	Percentages
Mild	24	32.00%
Minimal	26	34.67%
Moderate	25	33.33%

Among the study population, 24 (32.00%) of the participants had Mild depression, 26 (34.67%) of the participants had Minimal depression, and 25 (33.33%) of the participants had moderate depression. (Table 8 & Figure 5)

Figure 5: Pie chart of Depression severity in the study population (N=75)

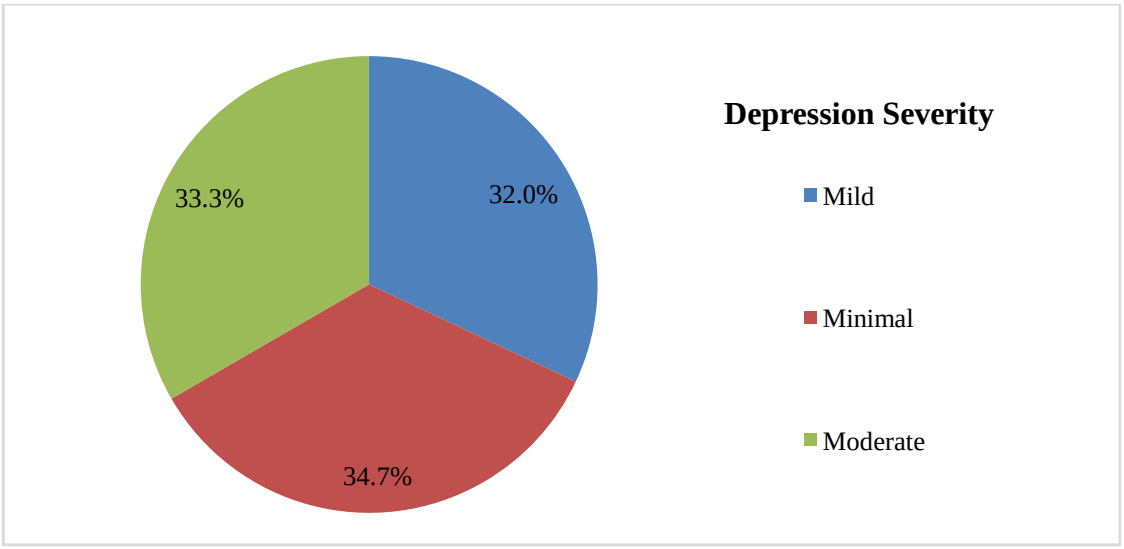


Table 9: Descriptive analysis of comorbidities in the study population (N=75)

Comorbidities	Frequency	Percentages
HTN	11	14.67%
No	64	85.33%

Among the study population in comorbidities, 11 (14.67%) of the participants had HTN. (Table 9 & Figure 6)

Figure 6: Bar chart of comorbidities in the study population (N=75)

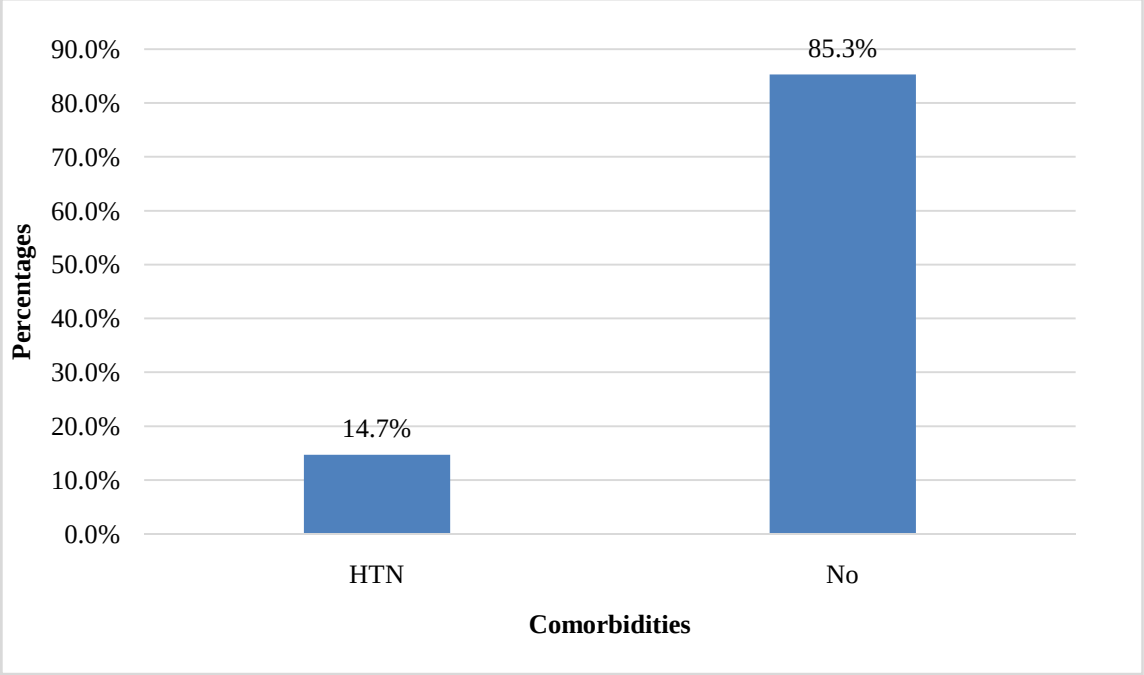


Table 10: Descriptive analysis of ART regimen in the study population (N=75)

ART Regimen	Frequency	Percentages
TLE	53	70.67%
ZLE	10	13.33%
Not on ART	12	16.00%

Among the study population in ART regimen, 53 (70.67%) of the participants had TLE, 10 (13.33%) of the participants had ZLE. (Table 10 & Figure 7)

Figure 7: Bar chart of ART regimen in the study population (N=75)

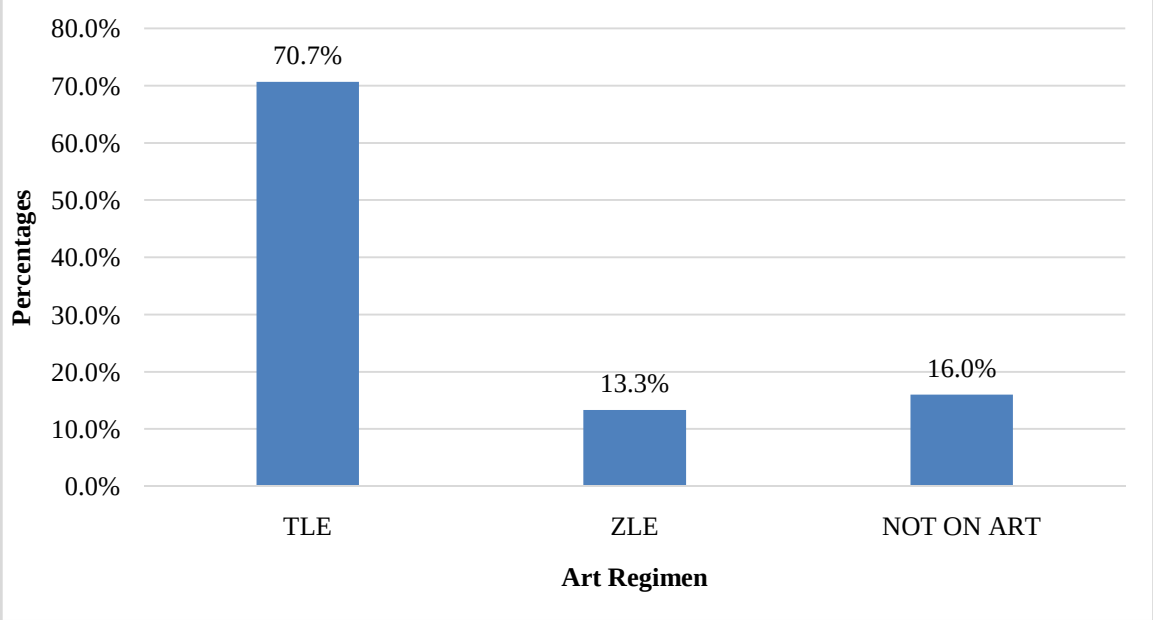


Table 11: Descriptive analysis of PHQ-9, Serum Free Testosterone, CD4 count, duration in months, ART regimen duration in study population (N=75)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
PHQ-9	7.13 ± 4.06	6.00	1.00	14.00	6.20	8.07
Serum Free Testosterone	8.62 ± 6.99	7.41	0.09	28.30	7.01	10.23
CD4 Count	349.75 ±240.52	268.00	17.00	854.00	294.41	405.08
Duration in Months	94.67 ± 36.01	96.00	12.00	192.00	85.60	103.73
ART Regimen Duration	4.76 ± 2.31	5.00	1.00	12.00	4.18	5.34

The mean PHQ-9 was 7.13 ± 4.06 in our study population, minimum PHQ-9 was 1, and the maximum PHQ-9 was 14 (95% CI 6.20 to 8.07).The mean SerumTestosterone was 8.62 ± 6.99 in the study population, minimum Serum Testosterone was 0.09, and maximum Serum Testosterone was 28.30 in the (95% CI 7.01 to 10.23).The mean CD4 Count was 349.75 ±240.52, and minimum CD4 Count was 17, and maximum CD4 Count was 854 in our study population (95% CI 294.41 to 405.08). The mean Duration in Months was 94.67 ± 36.01, minimum duration was 12, and the maximum duration was 192 in the study population (95% CI 85.60 to 103.73). The mean ART Regimen Duration was 4.76 ± 2.31, minimum duration was 1, and the maximum duration was 12 in the current study population (95% CI 4.18 to 5.34). (Table 11)

Table 12: Descriptive analysis of laboratory parameters in the study population (N=75)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
Haemoglobin	11.31 ± 2.49	11.00	7.40	17.50	10.74	11.89
Platelet	2.4 ± 1.26	1.98	0.52	6.93	2.11	2.69
Tlc	7.6 ± 3.19	7.50	1.28	15.80	6.87	8.34
Creatinine	1.03 ± 0.33	0.95	0.45	2.50	0.95	1.10

The mean Haemoglobin was 11.31 ± 2.49, and minimum haemoglobin was 7.40, and maximum haemoglobin was 17.50 in the study population (95% CI 10.74 to 11.89).The mean Platelet was 2.4 ± 1.26, and minimum platelet was 0.52, and maximum platelet was 6.93 in the study population (95% CI 2.11 to 2.69).The mean TLC was 7.6 ± 3.19 in the study population, minimum TLC was 1.28, and maximum TLC was 15.80(95% CI 6.87 to 8.34).The mean Creatinine was 1.03 ± 0.33, and minimum creatinine was 0.45, and maximum creatinine was 2.50 in the study population (95% CI 0.95 to 1.10). (Table 12)

Table 13: Descriptive analysis of bilirubin in the study population (N=75)

Bilirubin	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
Total Bilirubin	0.82 ± 0.51	0.80	0.13	2.77	0.70	0.94
Direct Bilirubin	0.33 ± 0.23	0.25	0.03	1.20	0.28	0.38

The mean Total Bilirubin was 0.82 ± 0.51, and minimum total bilirubin was 0.13, and maximum total bilirubin was 2.77 (95% CI 0.70 to 0.94).In the current study, the population mean Direct Bilirubin was 0.33 ± 0.23, and minimum direct bilirubin was 0.03, and maximum direct bilirubin was 1.20 (95% CI 0.28 to 0.38). (Table 13)

Table 14: Descriptive analysis of clinical parameters in the study population (N=75)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
SGOT	31.55 ± 22.47	25.00	10.00	126.00	26.38	36.72
SGPT	28.11 ± 21.24	21.00	5.00	138.00	23.22	32.99
Cholesterol	134.07 ± 34.11	135.00	69.00	262.00	126.22	141.92
LDL	58.23 ± 23.74	58.00	11.00	122.00	52.77	63.69
HDL	34.51 ± 16.07	32.00	12.00	117.00	30.81	38.20
Triglycerides	120.84 ± 41.79	110.00	46.00	280.00	111.22	130.46

The mean SGOT was 31.55 ± 22.47, and the minimum was 10, and the maximum was 126 in the study population (95% CI 26.38 to 36.72).The mean SGPT was 28.11 ± 21.24, and the minimum was 5, and the maximum was 138 in the study population (95% CI 23.22 to 32.99).

The mean Cholesterol was 134.07 ± 34.11, and the minimum was 69, and the maximum was 262 in the study population (95% CI 126.22 to 141.92). The mean LDL was 58.23 ± 23.74, and the minimum was 11, and the maximum was 122 in the study population (95% CI 52.77 to 63.69).The mean HDL was 34.51 ± 16.07, and the minimum was 12, and the maximum was 117 in the study population (95% CI 30.81 to 38.20).The mean Triglycerides was 120.84 ± 41.79 and minimum was 46, and the maximum was 280 in the study population (95% CI 111.22 to 130.46). (Table 14)

Table 15: Descriptive analysis of USG abdomen in the study population (N=75)

USG Abdomen	Frequency	Percentages
Mild hepatomegaly and splenomegaly	1	1.33%
Acalculous cholecystitis	2	2.67%
Acute pancreatitis	1	1.33%
Cholelithiasis	3	4.00%
Fatty liver	4	5.33%
Fatty liver,acute pancreatitis	1	1.33%
Fatty liver,enlarged prostate	2	2.67%
Fatty liver, right small kidney, grade 2 RPC	1	1.33%
Fatty liver, splenomegaly	3	4.00%
Grade 1 renal parenchymal changes	2	2.67%
Grade 1 RPC	6	8.00%
Hepatomegaly with fatty liver	1	1.33%
Hepatomegaly with fatty liver, grade 1 RPC	1	1.33%
Hepatosplenomegaly,cystitis,	4	5.33%
Mild splenomegaly	4	5.33%
Mild splenomegaly, mild ascites	3	4.00%
Mild hepatomegaly and acute pancreatitis	1	1.33%
Mild hepatomegaly and fatty infiltration	3	4.00%
Mild hepatomegaly and fatty infiltration, grade 1 RPC	1	1.33%
Mild hepatomegaly and splenomegaly	5	6.67%
Prostatomegaly	1	1.33%
No	25	33.33%

Among the study population in USG abdomen, 1 (1.33%) had Mild hepatomegaly, and splenomegaly, 2 (2.67%) had Acalculous cholecystitis, 1 (1.33%) had Acute pancreatitis, 3 (4%) had Cholelithiasis, 4 (5.33%) had Fatty liver, 1 (1.33%) had Fatty liver, acute pancreatitis, 2 (2.67%) had Fatty liver, enlarged prostate, 1 (1.33%) had

Fatty liver, right small kidney, grade 2 RPC, 3 (4%) had Fatty liver, splenomegaly, 2 (2.67%) had Grade 1 renal parenchymal changes, 6 (8%) had Grade 1 RPC, 1 (1.33%) had Hepatomegaly with fatty liver, 1 (1.33%) had Hepatomegaly with fatty liver, grade 1 RPC, 4 (5.33%) had Hepatosplenomegaly, cystitis, 4 (5.33%) had Mild splenomegaly, 3 (4%) had Mild splenomegaly, mild ascites, 1 (1.33%) had Mild hepatomegaly, and acute pancreatitis, 3 (4%) had Mild hepatomegaly, and fatty infiltration, 1 (1.33%) had Mild hepatomegaly, and fatty infiltration, grade 1 RPC, 5 (6.67%) had Mild hepatomegaly, and splenomegaly, 1 (1.33%) had Prostatomegaly. (Table 15)

Table 16: Descriptive analysis of C-XRY in the study population (N=75)

CXR	Frequency	Percentages
No	75	100.00%

Among the study population, there was no CXR. (Table 16)

Table 17: Comparison of age group across IIEF score group (N=75)

Age Group	IIEF score group			
	Moderate ED	Mild-Moderate ED	Mild ED	No ED
20-49Yrs (N=50)	3 (6%)	23 (46%)	21 (42%)	3 (6%)
>= 50Yrs (N=25)	1 (4%)	18 (72%)	6 (24%)	0 (0%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the age group 20-49yrs, 3 (6%) of the participants had Moderate ED, 23 (46%) of the participants had Mild-Moderate ED, 21 (42%) of the participants had Mild ED, and 3 (6%) of the participants had No ED. In the age group >= 50yrs, 1 (4%) of the participants had Moderate ED, 18 (72%) of the participants had Mild-Moderate ED, 6 (24%) of the participants had Mild ED. (Table 17 &Figure 8)

Figure 8: Cluster bar chart of comparison of age group across IIEF score group (N=75)

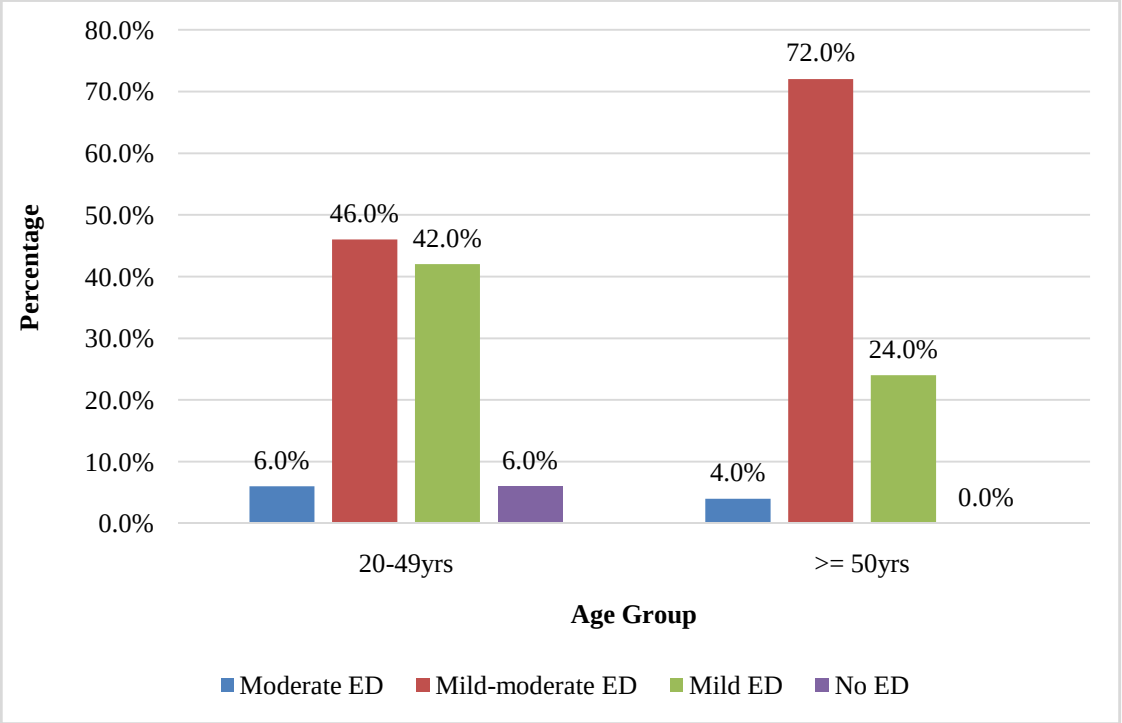


Table 18: Descriptive analysis of gender across IIEF score group (N=75)

Gender	IIEF score group			
	Moderate ED	**Mild-Moderate ED**	**Mild ED**	**No ED**
Male (N=75)	4 (5.33%)	41 (54.67%)	27 (36%)	3 (4%)

Among the study population, 4 (5.33%) of the male participants had Moderate ED, 41 (54.67%) of the male participants had Mild-Moderate ED, 27 (36%) of the male participants had Mild ED, and 3 (4%) of the male participants had No ED. (Table 18)

Table 19: Comparison of IIEF score across Depression severity (N=75)

Parameter	Depression Severity Median (IQR)			Kruskal Wallis test (P value)
	Mild (N=24)	**Minimal (N=26)**	**Moderate (N=25)**	
IIEF score	16 (14.25 to 18.75)	18 (16.75 to 20)	14 (12.50 to 15)	<0.001

Among the study population, IIEF score was 16 (IQR 14.25 to 18.75) for Mild depression, 18 (IQR 16.75 to 20) for Minimal depression and 14 (IQR 12.50 to 15) for moderate depression severity. The relation between depression severity and IIEF score was statistically significant (P value <0.001). (Table 19)

Table 20: Comparison of comorbidities across IIEF score group (N=75)

Comorbidities	IIEF score group			
	Moderate ED	**Mild-Moderate ED**	**Mild ED**	**No ED**
HTN (N=11)	1 (9.09%)	7 (63.64%)	3 (27.27%)	0 (0%)
No (N=64)	3 (4.69%)	34 (53.13%)	24 (37.5%)	3 (4.69%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the HTN comorbidities, 1 (9.09%) of the participants had Moderate ED, 7 (63.64%) of the participants had Mild-Moderate ED and 3 (27.27%) of the participants had Mild ED. (Table 20 & Figure 9)

Figure 9: Cluster bar chart of comparison of comorbidities across IIEF score group (N=75)

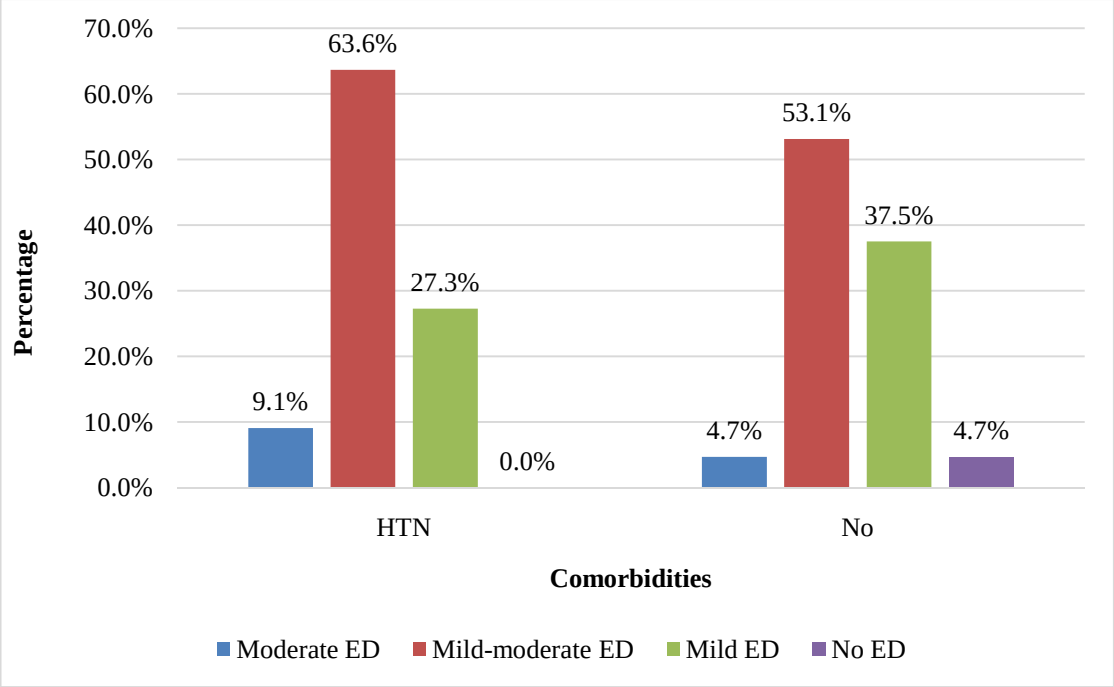


Table 21: Comparison of ART regimen across IIEF score group (N=75)

ART Regimen	IIEF score group			
	Moderate ED	Mild-Moderate ED	Mild ED	No ED
TLE (N=53)	3 (5.66%)	30 (56.6%)	19 (35.85%)	1 (1.89%)
ZLE (N=10)	1 (10%)	6 (60%)	3 (30%)	0 (0%)
Not on Art (N=12)	0 (0%)	5 (41.67%)	5 (41.67%)	2 (16.67%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the TLE ART regimen, 3 (5.66%) of the participants had Moderate ED, 30 (56.6%) of the participants had Mild-Moderate ED, 19 (35.85%) of the participants had Mild ED, and 1 (1.89%) of the participants had no ED. In the ZLE ART regimen, 1 (10%) of the participants had Moderate ED, 6 (60%) of the participants had Mild-Moderate ED and 3 (30%) of the participants had Mild ED. (Table 21 & Figure 10)

Figure 10: Cluster bar chart of comparison of ART regimen across IIEF score group (N=75)

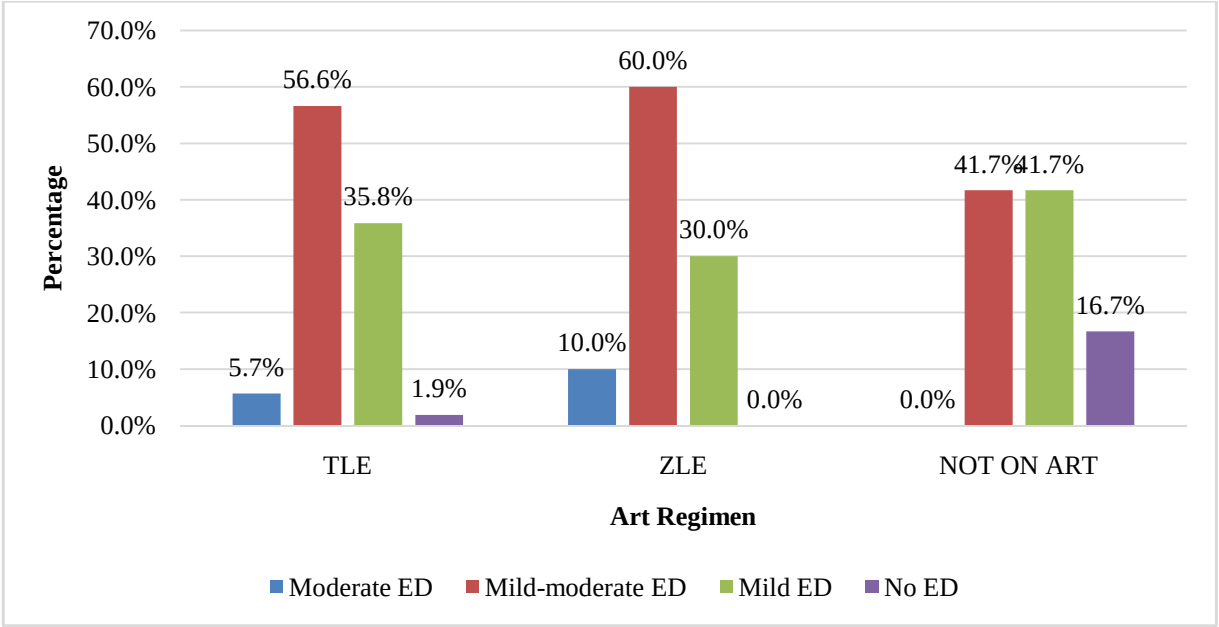


Table 22: Comparison of serum Testosterone group across IIEF score group (N=75)

Serum testosterone	IIEF score group			
	Moderate ED	Mild-Moderate ED	Mild ED	No ED
Normal (N=34)	0 (0%)	11 (32.35%)	20 (58.82%)	3 (8.82%)
Abnormal (N=41)	4 (9.76%)	30 (73.17%)	7 (17.07%)	0 (0%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the normal serum Testosterone, 11 (32.35%) of the participants had Mild-Moderate ED, 20 (58.82%) of the participants had Mild ED, and 3 (8.82%) of the participants had no ED. In the abnormal serum Testosterone, 4 (9.76%) of the participants had Moderate ED, 30 (73.17%) of the participants had Mild-Moderate ED and 7 (17.07%) of the participants had Mild ED. (Table 22 & Figure 11)

Figure 11: Cluster bar chart of comparison of serum Testosterone group across IIEF score group (N=75)

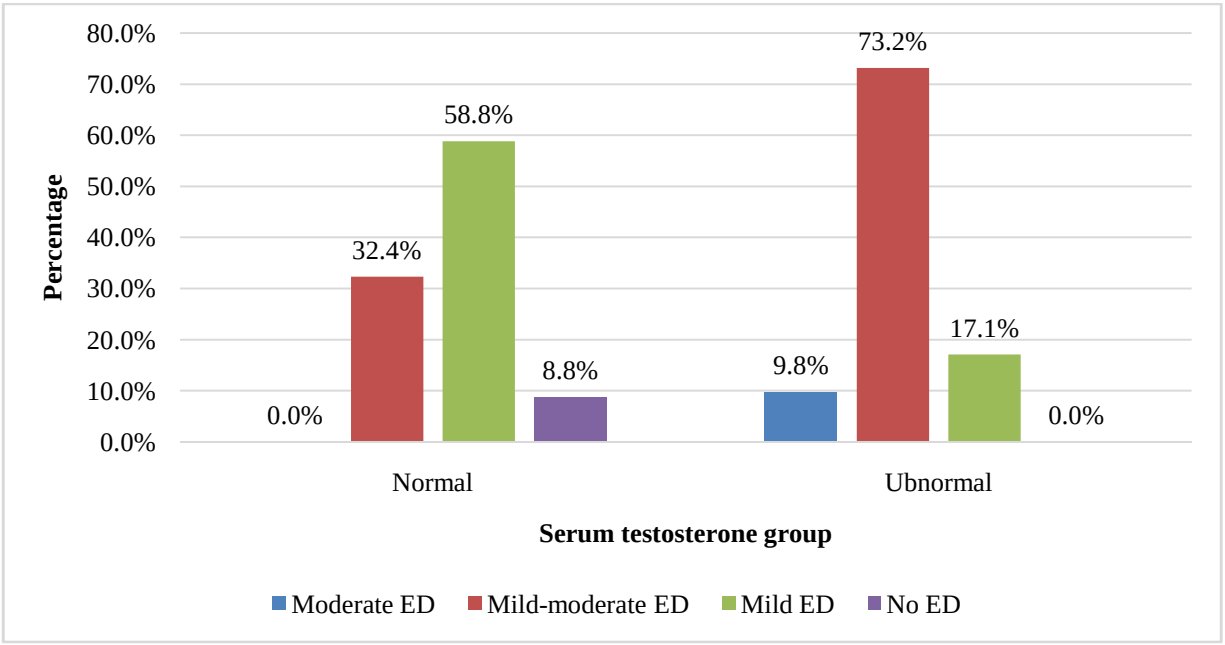


Table 23: Comparison of Hb group across IIEF score group (N=75)

Haemoglobin group	IIEF score group			
	Moderate ED	Mild-Moderate ED	Mild ED	No ED
Anemia (N=40)	4 (10%)	25 (62.5%)	10 (25%)	1 (2.5%)
Normal (N=35)	0 (0%)	16 (45.71%)	17 (48.57%)	2 (5.71%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the Anemia patients, 4 (10%) of the participants had Moderate ED, 25 (62.5%) of the participants had Mild-Moderate ED, 10 (25%) of the participants had Mild ED, and 1 (2.5%) of the participants had no ED. (Table 23 & Figure 12)

Figure 12: Cluster bar chart of comparison of Hb group across IIEF score group (N=75)

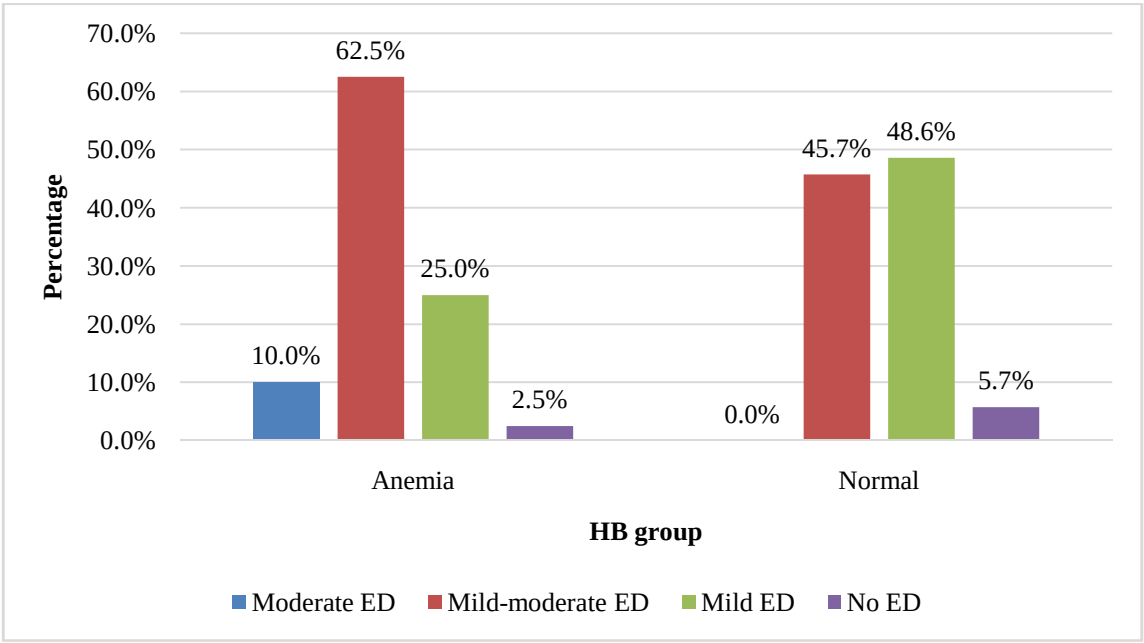


Table 24: Correlation between Erectile Dysfunction and Serum Testosterone in the study population (N=75)

Parameter	Spearman Correlation (rs)	P value
Serum testosterone	0.680	<0.001

As p value <0.001 which is statistically significant and moderate positive correlation between Serum Testosterone and Erectile dysfunctionin current study population (rs Value: 0.680, P value: <0.001) (Table 24 & Figure 13)

Figure 13: Scatter plot of Erectile Dysfunction and serum Testosterone in the study population (N=75)

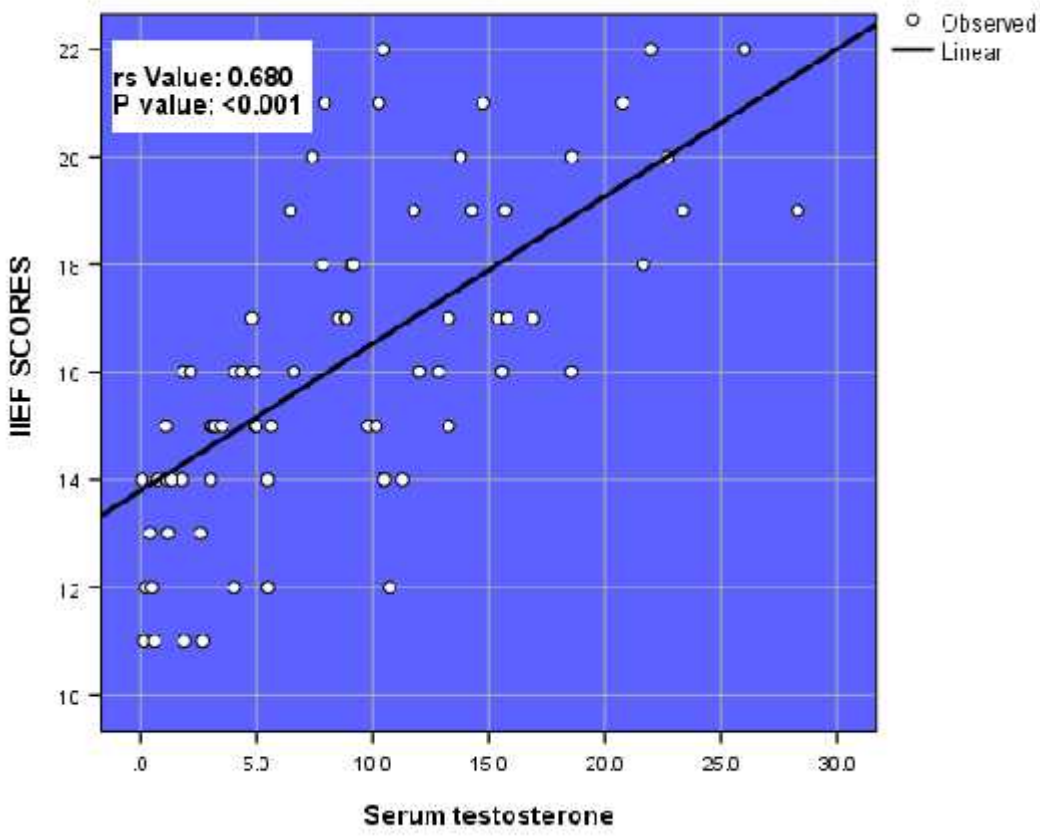


Table 25: Correlation between Erectile Dysfunction and CD4 count when serum Testosterone is normal in the Age group 20-49yrs (N=21)

Parameter	Spearman Correlation (rs)	P value
CD4 count	0.316	0.163

There was a weak positive correlation between CD4 count and Erectile dysfunctionwhen serum Testosterone was normal in the age group 20-49yrs(rs Value: 0.316, P value: 0.163). (Table 25 & Figure 14) which is not significant.

Figure 14: Scatter plot of Erectile Dysfunction and CD4 count when serum Testosterone is normal in the age group 20-49yrs (N=21)

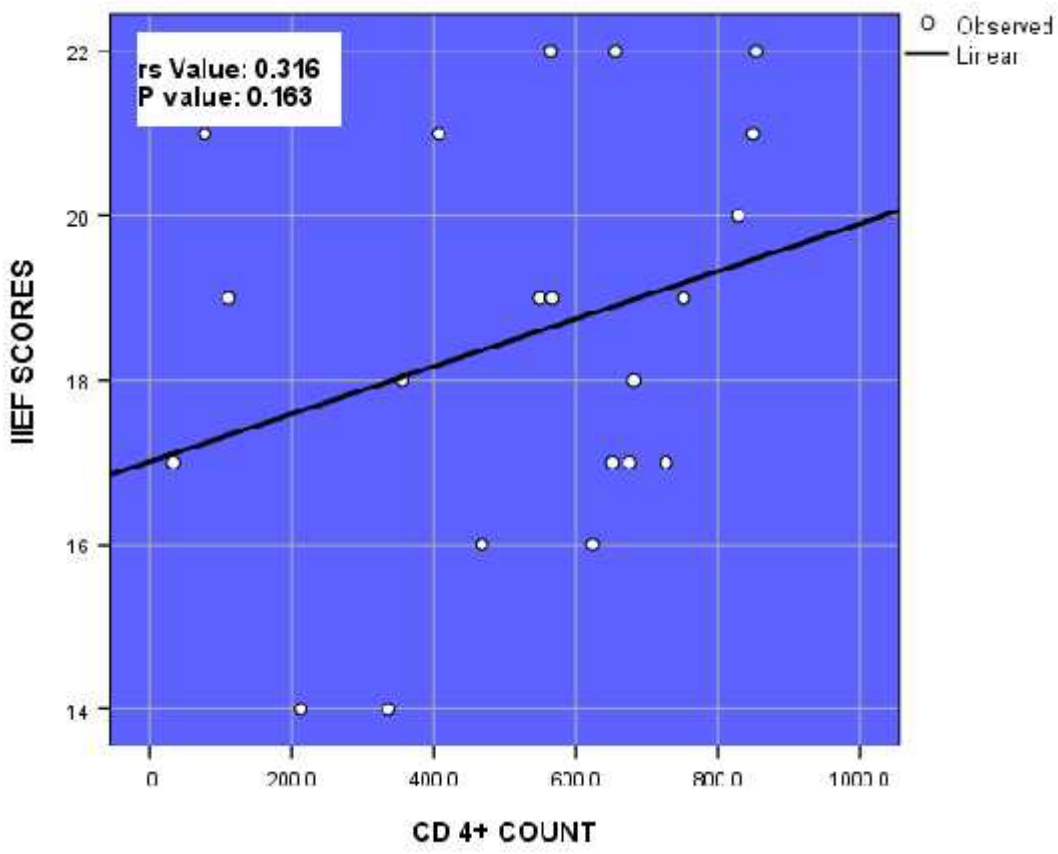


Table 26: Correlation between Erectile Dysfunction and CD 4+ count when serum Testosterone is abnormal in the age group 20-49yrs (N=29)

Parameter	Spearman Correlation (rs)	P value
CD 4+ count	0.482	0.008

There was a weak positive correlation between CD4 count and Erectile dysfunctionwhen serum Testosterone was abnormal in the age group 20-49yrs(rs Value: 0.482, P value: 0.008) (Table 26 & Figure 15)

Figure 15: Scatter plot of Erectile Dysfunction and CD4 count when serum Testosterone is abnormal in the age group 20-49yrs (N=29)

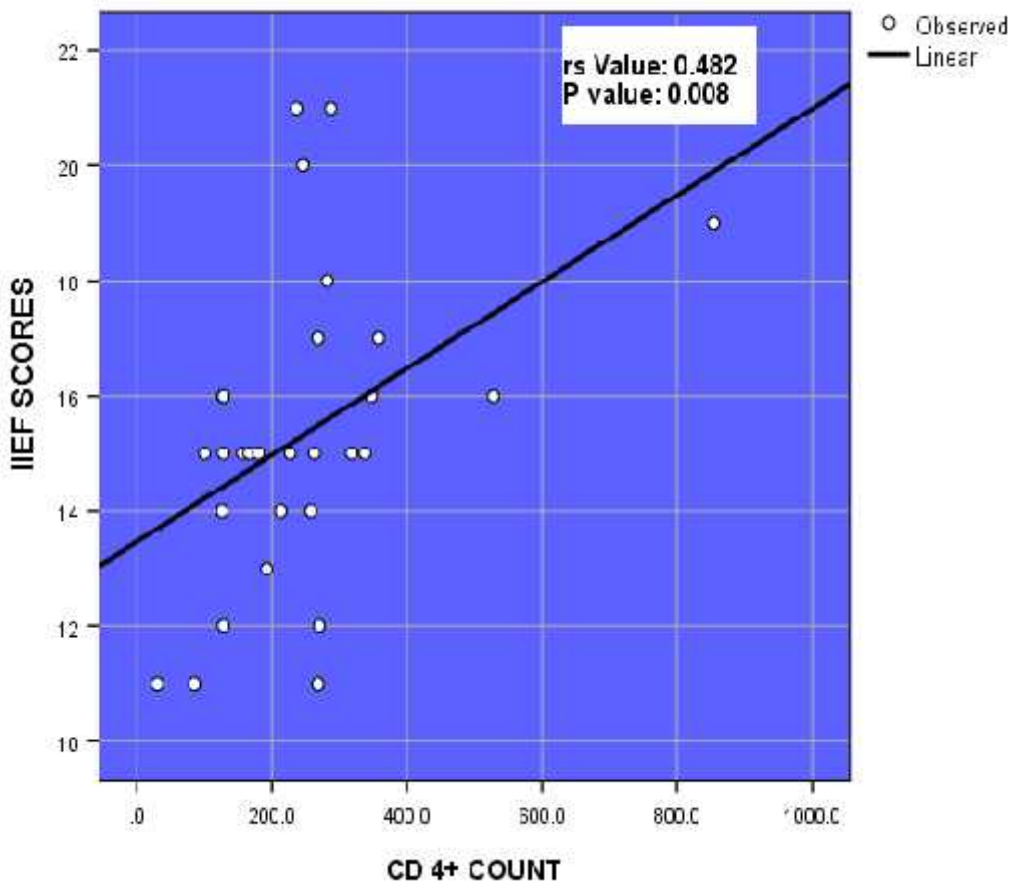


Table 27: Correlation between Erectile Dysfunction and CD4 count when serum Testosterone is normal in the age group > or =50 (N=13)

Parameter	Spearman Correlation (rs)	P value
CD 4 count	0.155	0.612

There was a weak positive correlation between CD4 count and Erectile dysfunctionwhen serum Testosterone was normal in the age group > or =50yrs (rs Value: 0.155, P value: 0.612) (Table 27 & Figure 16)

Figure 16: Scatter plot of Erectile Dysfunction and CD4 count when serum Testosterone is normal in the age group > or =50 (N=13)

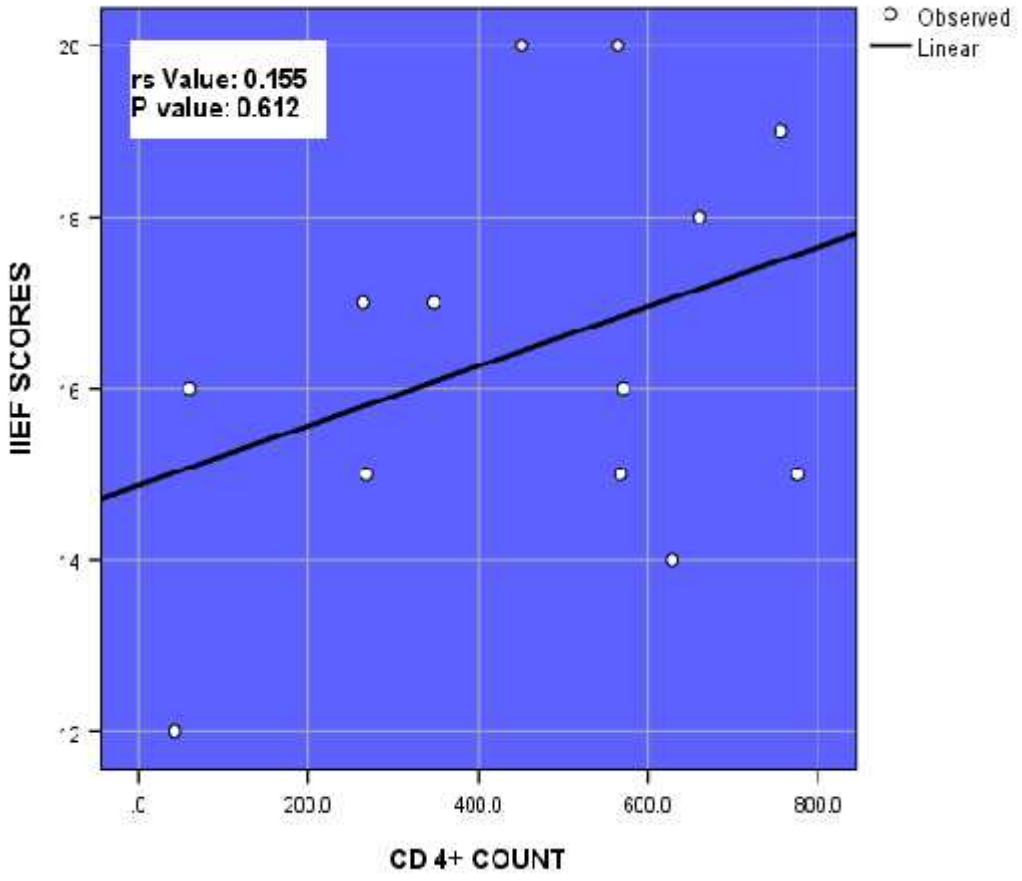


Table 28: Correlation between Erectile Dysfunction and CD4 count when serum Testosterone is abnormal in the age group > or =50 (N=12)

Parameter	Spearman Correlation (rs)	P value
CD4 count	0.013	0.969

There was a weak positive correlation between CD 4 count and Erectile dysfunctionwhen serum Testosterone was abnormal in the age group > or =50yrs (rs Value: 0.013, P value: 0.969) (Table 28 & Figure 17)

Figure 17: Scatter plot of Erectile dysfunction and CD4 count when serum Testosterone is abnormal in the age group > or =50 (N=12)

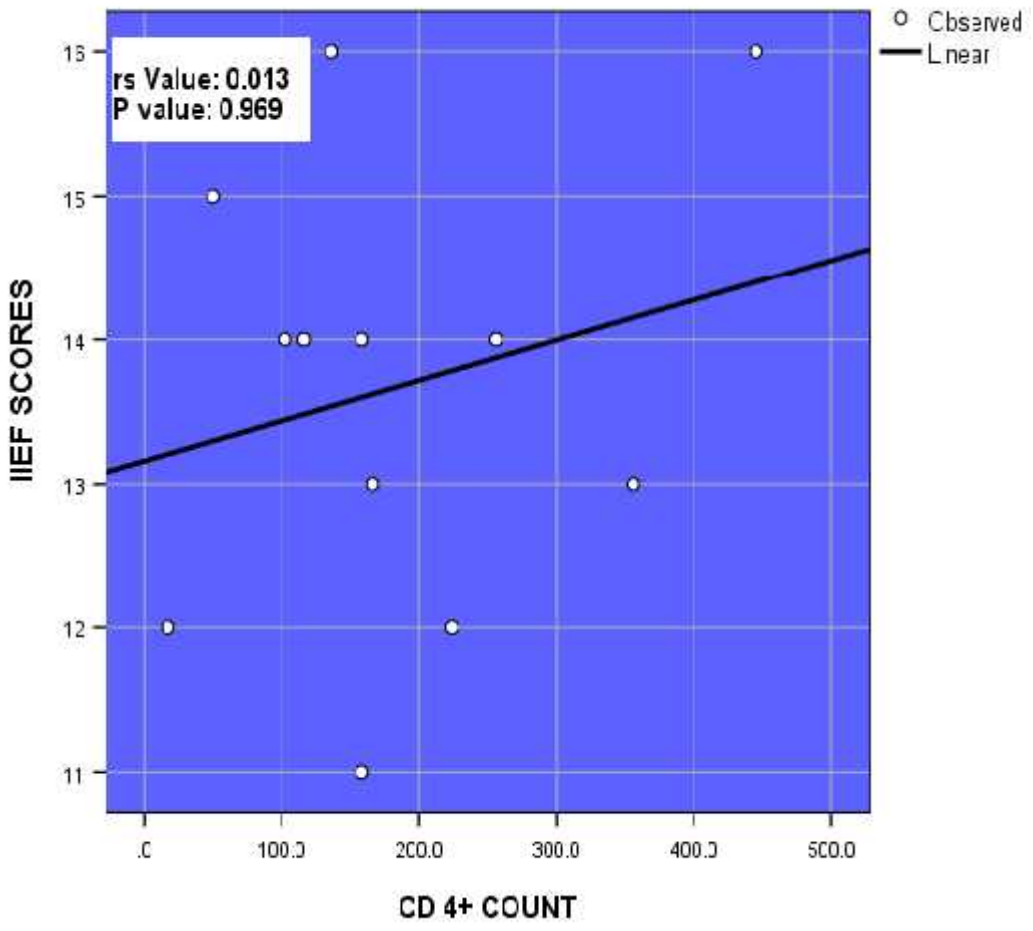


Table 29: Correlation between Erectile Dysfunction and ART regimen duration in normal serum Testosterone (N=34)

Parameter	Spearman Correlation (rs)	P value
ART regimen duration	-0.458	0.014

There was a weak negative correlation between ART regimen duration and Erectile dysfunctionwhen serum Testosterone was normal in the study population(rs Value: -0.458, P value: 0.014) (Table 29 & Figure 18)

Figure 18: Scatter plot of Erectile Dysfunction and ART regimen duration in normal serum Testosterone (N=34)

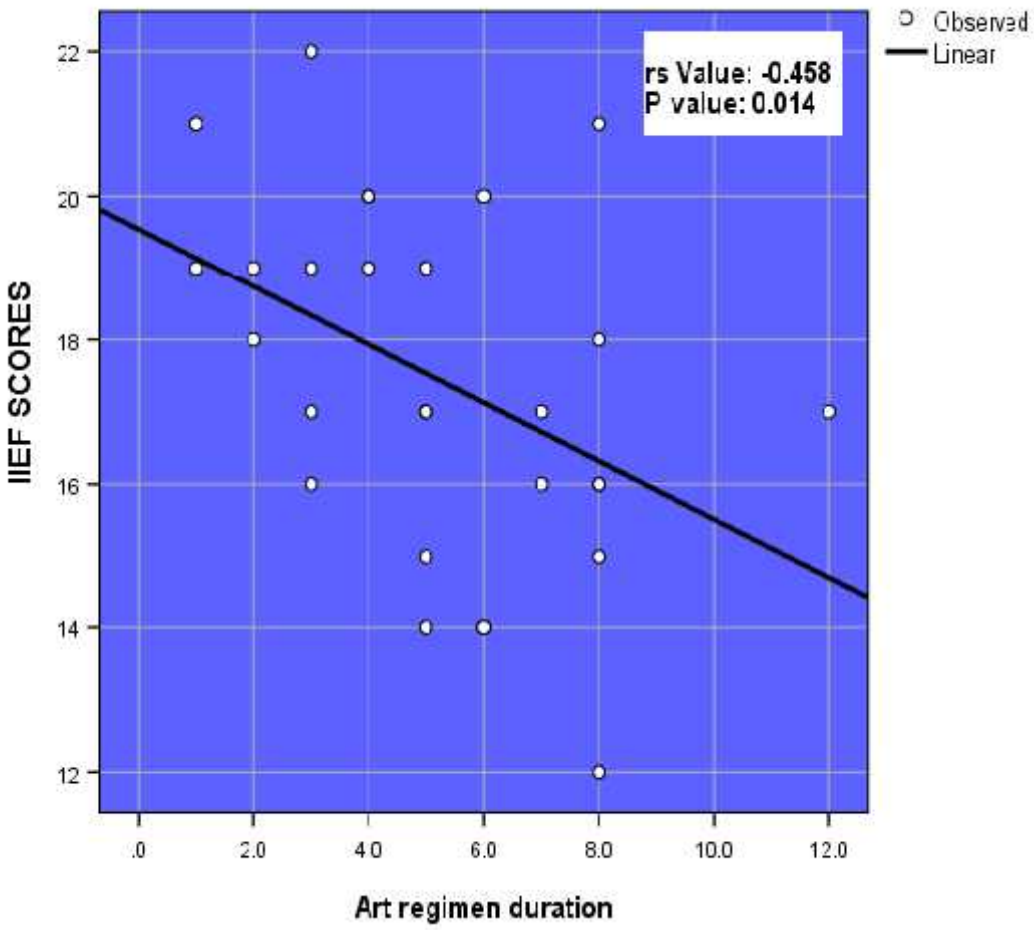


Table 30: Correlation between Erectile Dysfunction and ART regimen duration in abnormal serum Testosterone (N=41)

Parameter	Spearman Correlation (rs)	P value
ART regimen duration	-0.288	0.093

There was a weak negative correlation between ART regimen duration and Erectile dysfunctionwhen serum Testosterone was abnormal in the study population(rs Value: -0.288, P value: 0.093) (Table 30 & Figure 19)

Figure 19: Scatter plot of Erectile Dysfunction and ART regimen duration in abnormal serum Testosterone (N=41)

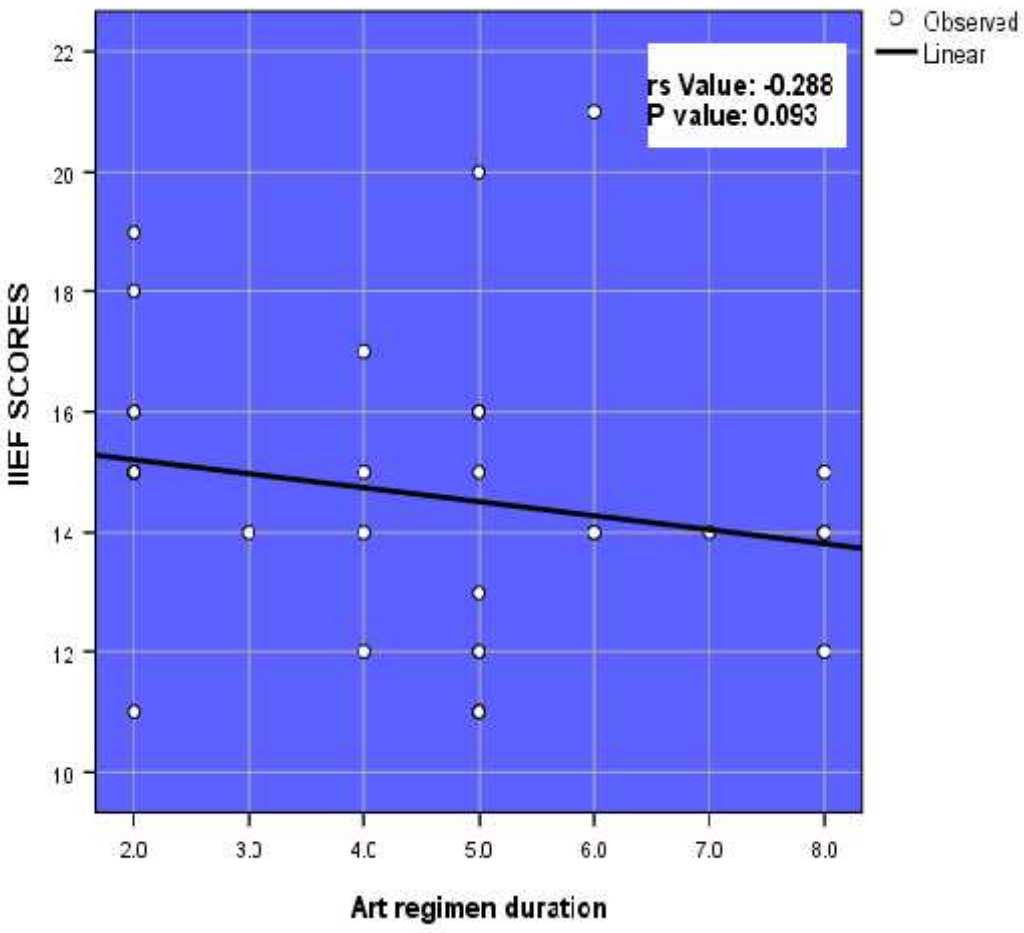


Table 31: Comparison of ART regimen across IIEF score group in serum Testosterone (normal) (N=34)

Art Regimen	IIEF score group		
	Mild-Moderate ED	Mild ED	No ED
TLE (N=25)	9 (36%)	15 (60%)	1 (4%)
ZLE (N=3)	1 (33.33%)	2 (66.67%)	0 (0%)
Not on ART(N=6)	1 (16.67%)	3 (50%)	2 (33.33%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the TLE ART regimen when serum testosterone was normal, 9 (36%) of the participants had Mild-Moderate ED, 15 (60%) of the participants had Mild ED and 1 (4%) of the participants had no ED. In the ZLE ART regimen, 1 (33.33%) of the participants had Mild-Moderate ED, 2 (66.67%) of the participants had Mild ED. (Table 31 & Figure 20)

Figure 20: Staked bar chart of comparison of ART regimen across IIEF score group in serum Testosterone (normal) (N=34)

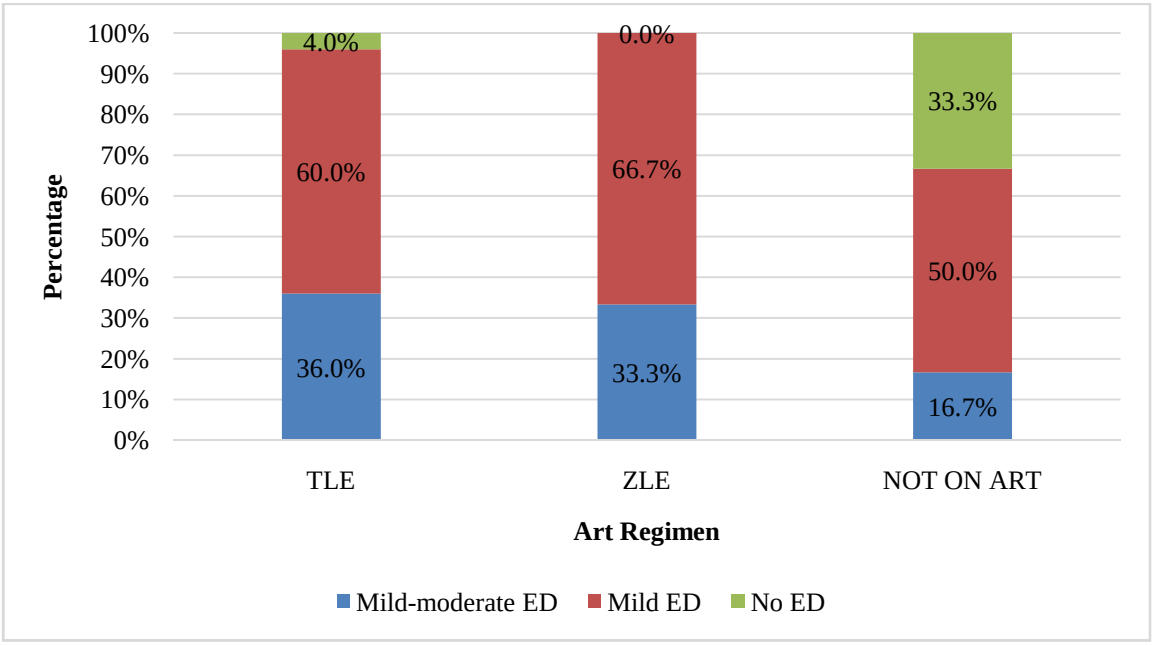


Table 32: Comparison of ART regimen across IIEF score group in serum Testosterone (abnormal) (N=41)

Art Regimen	IIEF score group		
	Moderate ED	**Mild-Moderate ED**	**Mild ED**
TLE (N=28)	3 (10.71%)	21 (75%)	4 (14.29%)
ZLE (N=7)	1 (14.29%)	5 (71.43%)	1 (14.29%)
Not on ART (N=6)	0 (0%)	4 (66.67%)	2 (33.33%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the TLE art regimen when serum testosterone was abnormal, 3 (10.71%) of the participants had Moderate ED, 21 (75%) of the participants had Mild- Moderate ED and 4 (14.29%) of the participants had Mild ED. In the ZLE art regimen, 1 (14.29%) of the participants had Moderate ED, 5 (71.43%) of the participants had Mild-Moderate ED, 1 (14.29%) of the participants had Mild ED. (Table 32 & Figure 21)

Figure 21: Staked bar chart of comparison of ART regimen across the IIEF score group in serum Testosterone (abnormal) (N=41)

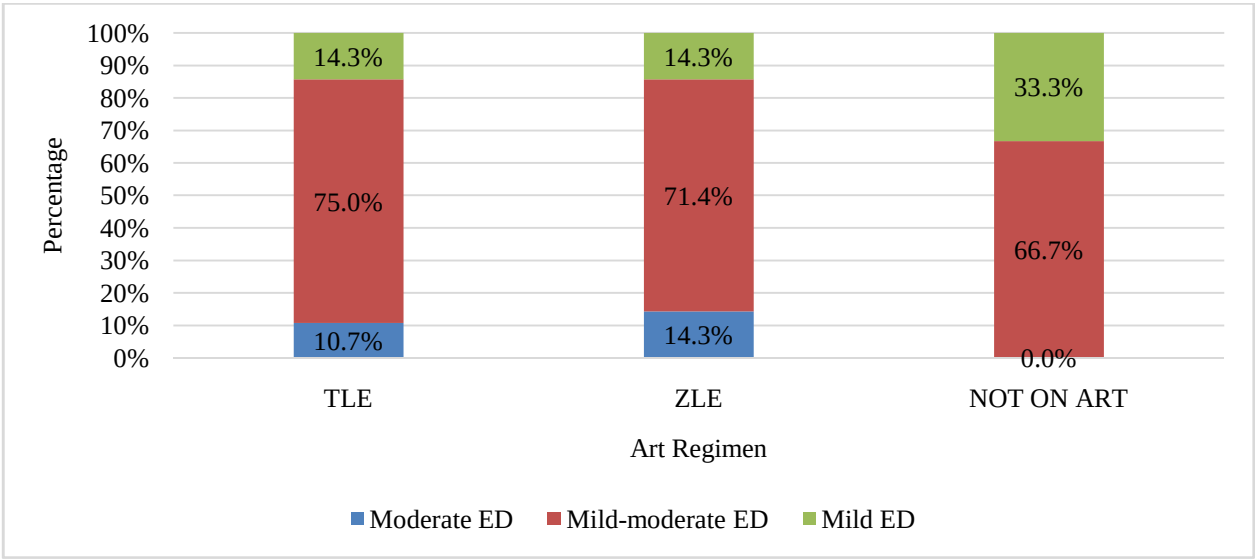


Table 33: Correlation between Erectile Dysfunction and age in normal serum Testosterone (N=34)

Parameter	Spearman Correlation (rs)	P value
Age	-0.568	<0.001

There was a moderate negative correlation between Age and Erectile dysfunctionwhen serum Testosterone was normal in the study population(rs Value: -0.568, P value: <0.001) (Table 33 & Figure 22)

Figure 22: Scatter plot of Erectile Dysfunction and age in normal serum Testosterone (N=34)

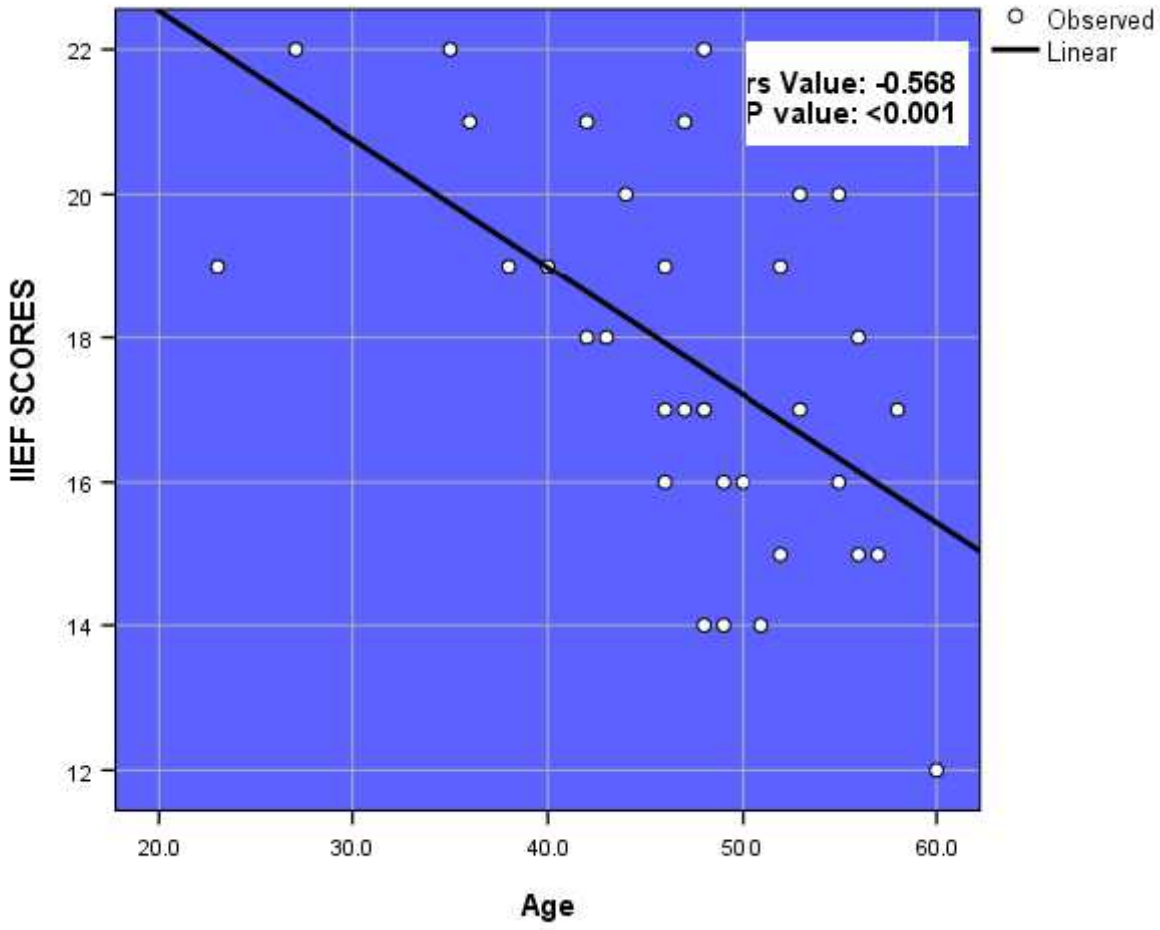


Table 34: Correlation between Erectile Dysfunction and age in abnormal serum Testosterone (N=41)

Parameter	Spearman Correlation (rs)	P value
Age	-0.459	0.003

There was a weak negative correlation between Age and Erectile dysfunctionwhen serum Testosterone was abnormal in the study population(rs Value: -0.459, P value: 0.003) (Table 34 & Figure 23)

Figure 23: Scatter plot of Erectile Dysfunction and age in abnormal serum Testosterone (N=41)

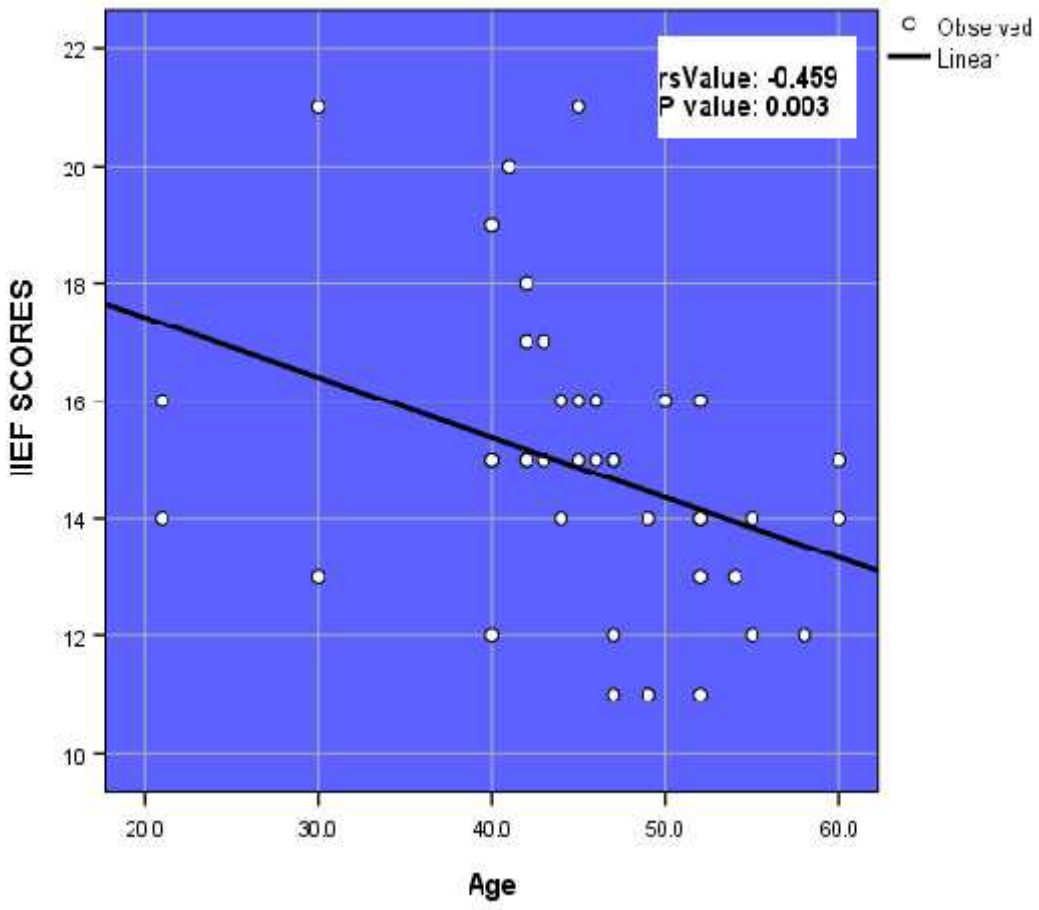


Table 35: Comparison of Depression severity across IIEF score group in serum Testosterone (normal) (N=34)

Depression Severity	IIEF score group		
	Mild-Moderate ED	Mild ED	No ED
Mild (N=12)	6 (50%)	5 (41.67%)	1 (8.33%)
Minimal (N=20)	3 (15%)	15 (75%)	2 (10%)
Moderate (N=2)	2 (100%)	0 (0%)	0 (0%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the mild depression severity when serum testosterone was normal, 6 (50%) of the participants had Mild-Moderate ED, 5 (41.67%) of the participants had Mild ED and 1 (8.33%) of the participants had No ED. In the minimal depression severity, 3 (15%) of the participants had Mild-Moderate ED, 15 (75%) of the participants had Mild ED, 2 (10%) of the participants had No ED. In the moderate depression severity, 2 (100%) of the participants had Mild-Moderate ED. (Table 35 & Figure 24)

Figure 24: Staked bar chart of comparison of Depression severity across IIEF score group in serum Testosterone (normal) (N=34)

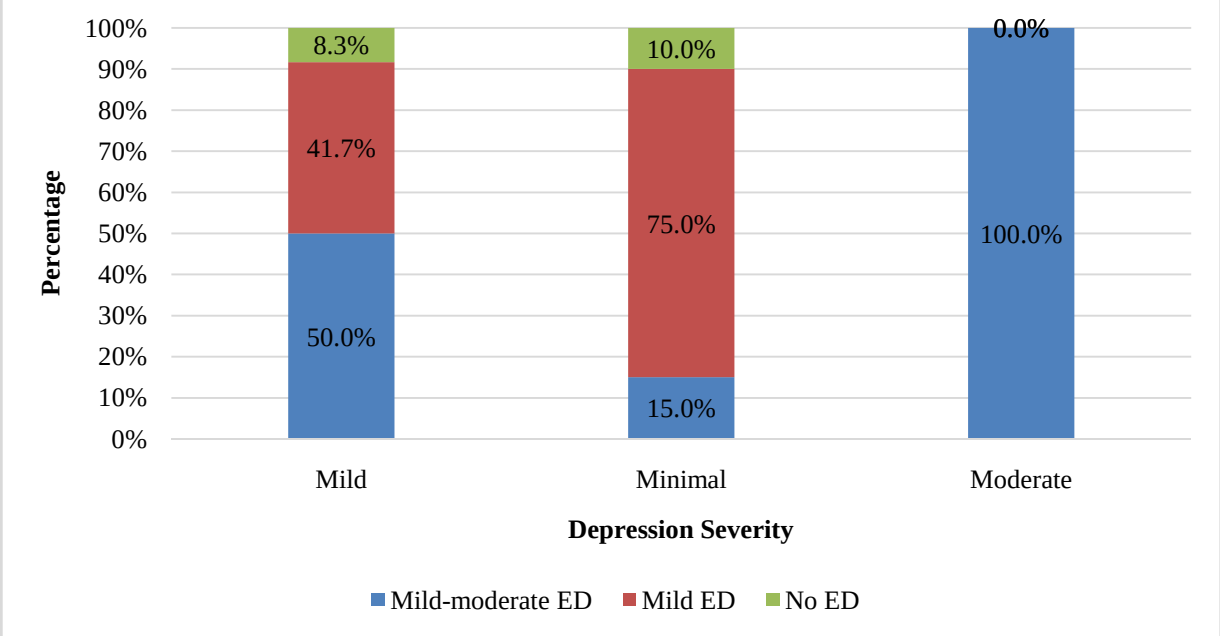


Table 36: Comparison of Depression severity across IIEF score group in serum Testosterone (abnormal) (N=41)

Depression Severity	IIEF score group		
	Moderate ED	**Mild-Moderate ED**	**Mild ED**
Mild (N=12)	0 (0%)	9 (75%)	3 (25%)
Minimal (N=6)	1 (16.67%)	2 (33.33%)	3 (50%)
Moderate (N=23)	3 (13.04%)	19 (82.61%)	1 (4.35%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the mild depression severity when serum testosterone was abnormal, 9 (75%) of the participants had Mild-Moderate ED, 3 (25%) of the participants had Mild ED. In the minimal depression severity, 1 (16.67%) of the participants had Moderate ED, 2 (33.33%) of the participants had Mild-Moderate ED, 3 (50%) of the participants had Mild ED. In the moderate depression severity, 3 (13.04%) of the participants had Moderate ED, 19 (82.61%) of the participants had Mild-Moderate ED and 1 (4.35%) of the participants had Mild ED. (Table 36 & Figure 25)

Figure 25: Staked bar chart of comparison of Depression severity across IIEF score group in serum Testosterone (abnormal) (N=41)

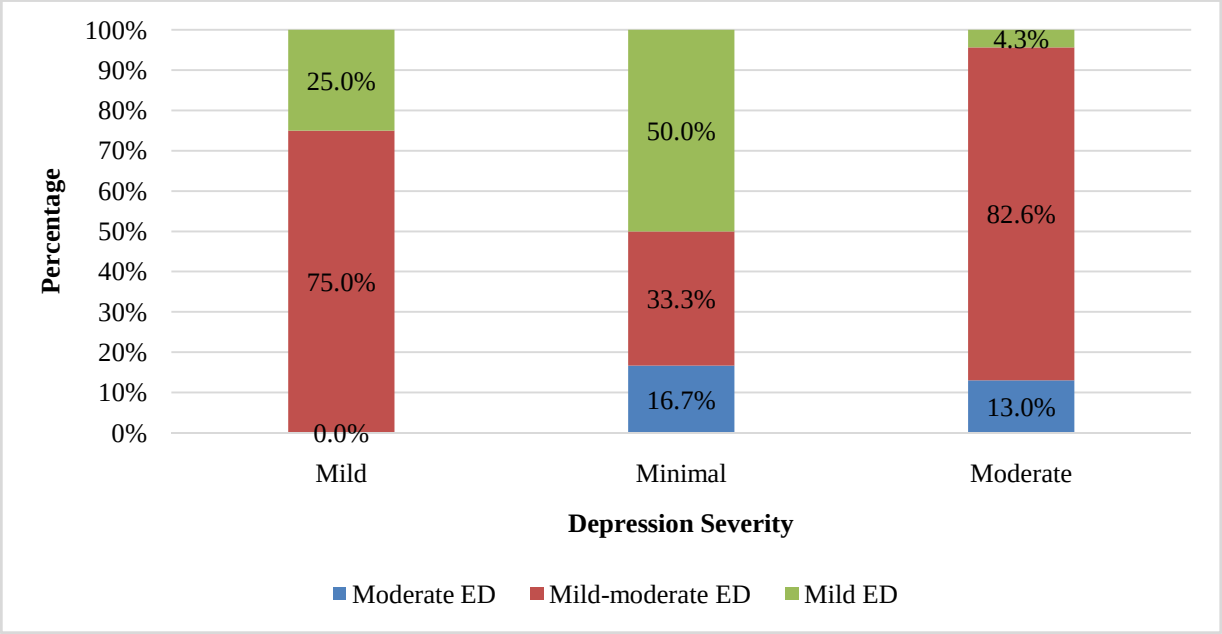


Table 37: Comparison of Hb group across IIEF score in serum Testosterone (normal) (N=34)

Haemoglobin	IIEF score group			Chi square	P value
	Mild-Moderate ED	Mild ED	No ED		
Anemia (N=12)	4 (33.33%)	7 (58.33%)	1 (8.33%)	0.011	0.994
Normal (N=22)	7 (31.82%)	13 (59.09%)	2 (9.09%)		

There was no statistically difference between hemoglobin and IIEF score groupwhen serum testosterone is normal in the study population for the P value of 0.994. (Table 37 & Figure 26)

Figure 26: Staked bar chart of comparison of HB group across IIEF score in serum Testosterone (normal) (N=34)

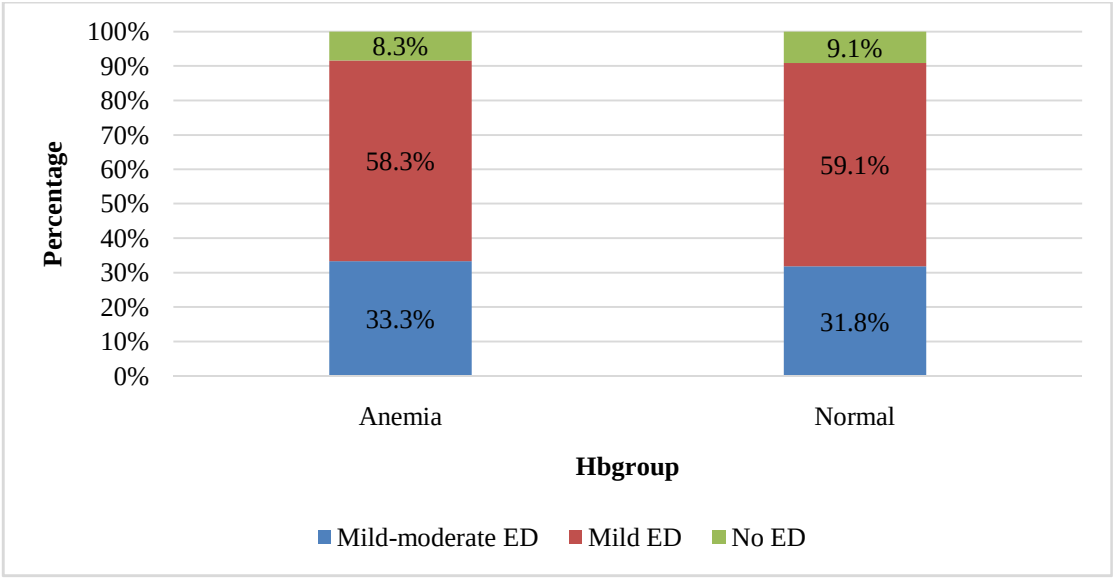


Table 38: Comparison of Hb group across IIEF score in serum Testosterone (abnormal)(N=41)

Haemoglobin	IIEF score group		
	Moderate ED	Mild-Moderate ED	Mild ED
Anemia (N=28)	4 (14.29%)	21 (75%)	3 (10.71%)
Normal (N=13)	0 (0%)	9 (69.23%)	4 (30.77%)

*No statistical test was applied- due to 0 subjects in the cells

Among the study population in the Anemia patients when serum testosterone is abnormal, 4 (14.29%) of the participants had Moderate ED, 21 (75%) of the participants had Mild-Moderate ED and 3 (10.71%) of the participants had Mild ED. (Table 38 & Figure 27)

Figure 27: Staked bar chart of comparison of Hb group across IIEF score in serum Testosterone (abnormal)(N=41)

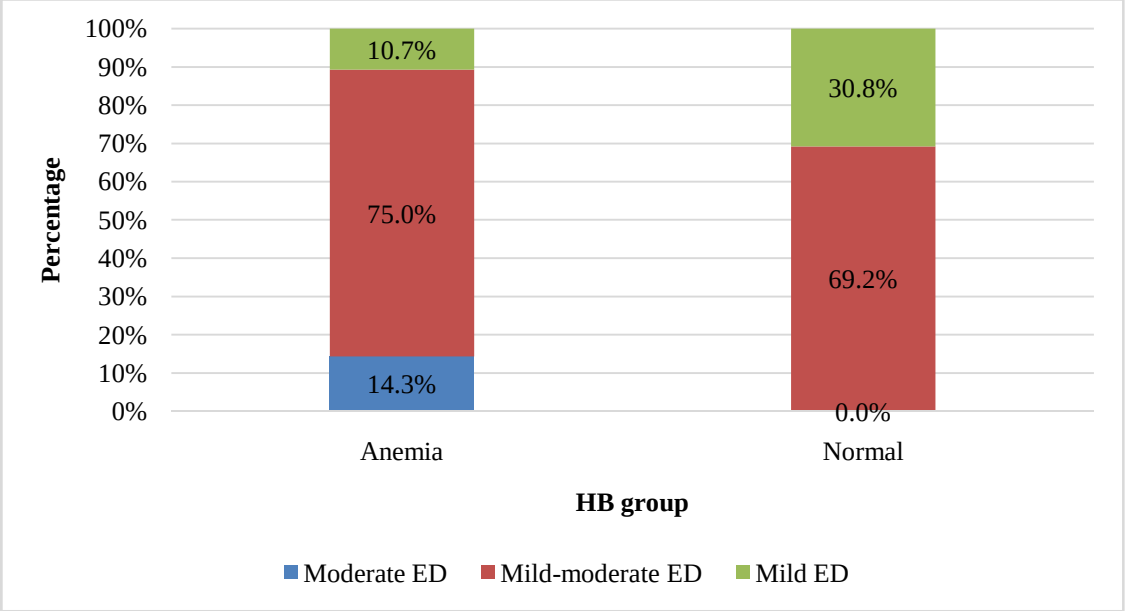


Table 39: Correlation between Erectile Dysfunction and BMI in normal serum Testosterone (N=34)

Parameter	Spearman Correlation (rs)	P value
BMI	0.037	0.834

There was a weak positive correlation between BMI and Erectile dysfunctionwhen serum testosterone was normal in the study population(rs Value: 0.037, P value: 0.834) (Table 39 & Figure 28)

Figure 28: Scatter plot of Erectile Dysfunction and BMI in normal serum Testosterone (N=34)

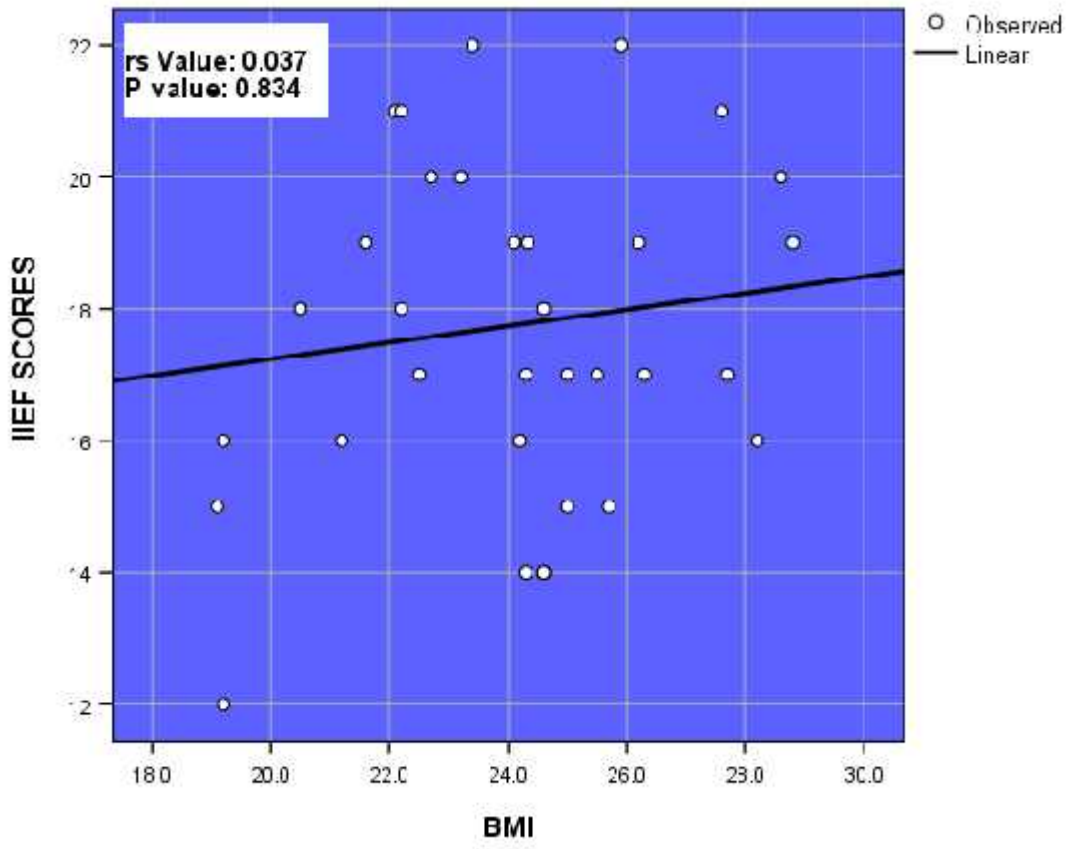


Table 40: Correlation between Erectile dysfunction and BMI in abnormal serum Testosterone (N=41)

Parameter	Spearman Correlation (rs)	P value
BMI	0.097	0.546

Here we got a weak positive correlation between BMI and Erectile dysfunctionwhen serum testosterone was abnormal in the study population(rs Value: 0.097, P value: 0.546) (Table 40 & Figure 29)

Figure 29: Scatter plot of Erectile Dysfunction and BMI in abnormal serum Testosterone (N=41)

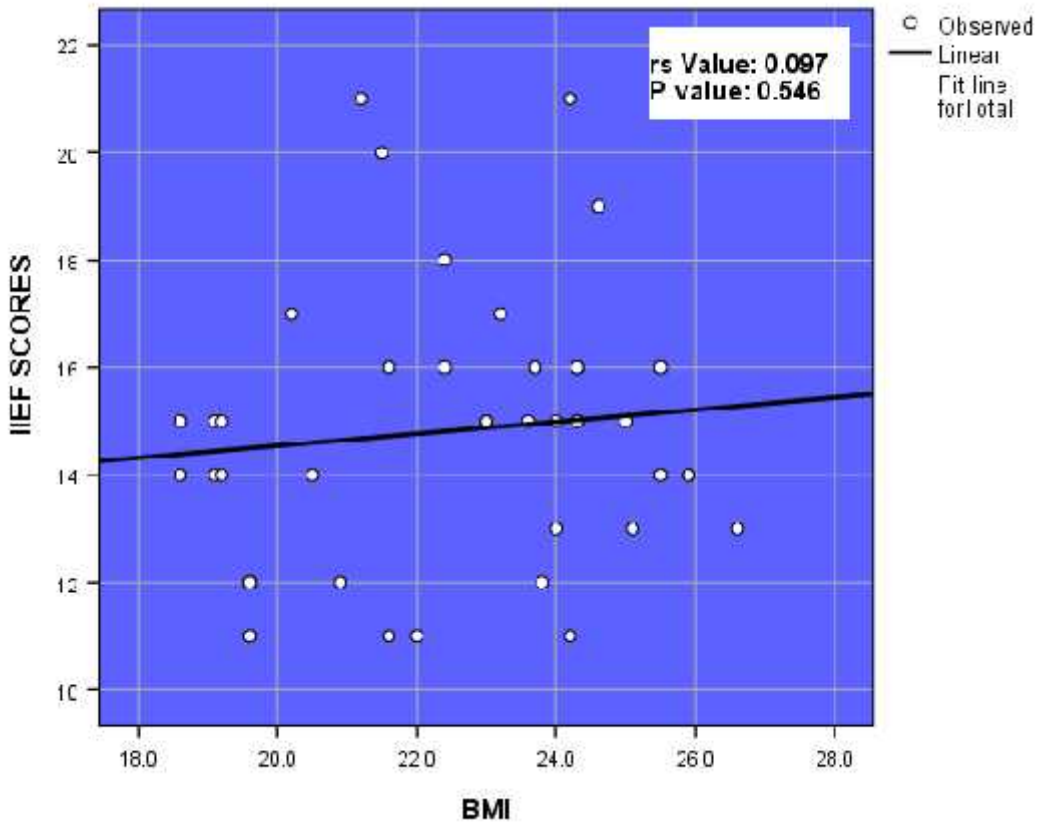


Table 41: Comparison of age group across IIEF score in serum Testosterone (normal) (N=34)

Age Group	IIEF score group		
	Mild-Moderate ED	Mild ED	No ED
20-49Yrs (N=21)	4 (19.05%)	14 (66.67%)	3 (14.29%)
>= 50Yrs (N=13)	7 (53.85%)	6 (46.15%)	0 (0%)

*No statistical test was applied due to 0 subjects in the cells.

Among the age group 20-49Yrs, 4 (19.05%) of the participants had Mild-Moderate ED, 14 (66.67%) of the participants had Mild ED, and 3 (14.29%) of the participants had No ED. Among the age group, more than 50Yrs, 7 (53.85%) of the participants had Mild-Moderate ED, 6 (46.15%) of the participants had Mild ED. (Table 41 & Figure 30)

Figure 30: Staked bar chart of comparison of age group across IIEF score in serum Testosterone (normal) (N=34)

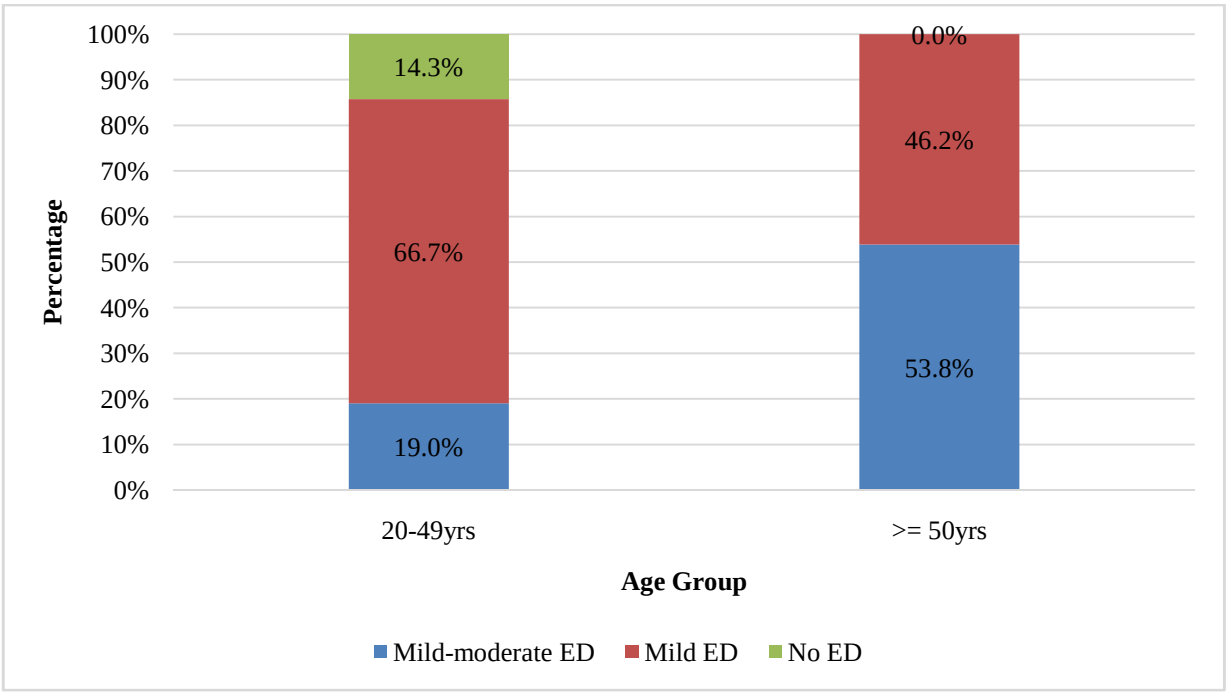


Table 42: Comparison of age group across IIEF score in serum Testosterone (abnormal) (N=41)

Age Group	IIEF score group		
	Moderate ED	Mild-Moderate ED	Mild ED
20-49Yrs (N=29)	3 (10.34%)	19 (65.52%)	7 (24.14%)
>= 50Yrs (N=12)	1 (8.33%)	11 (91.67%)	0 (0%)

*No statistical test was applied due to 0 subjects in the cells.

Among the current study population in age group 20-49Yrs when serum testosterone is abnormal, 3 (10.34%) of the participants had Moderate ED, 19 (65.52%) of the participants had Mild-Moderate ED and 7 (24.14%) of the participants had Mild ED. In the age group, more than 50Yrs, 1 (8.33%) of the participants had Moderate ED, 11 (91.67%) of the participants had Mild-Moderate ED. (Table 42 & Figure 31)

Figure 31: Staked bar chart of comparison of age group across IIEF score in serum Testosterone (abnormal) (N=41)

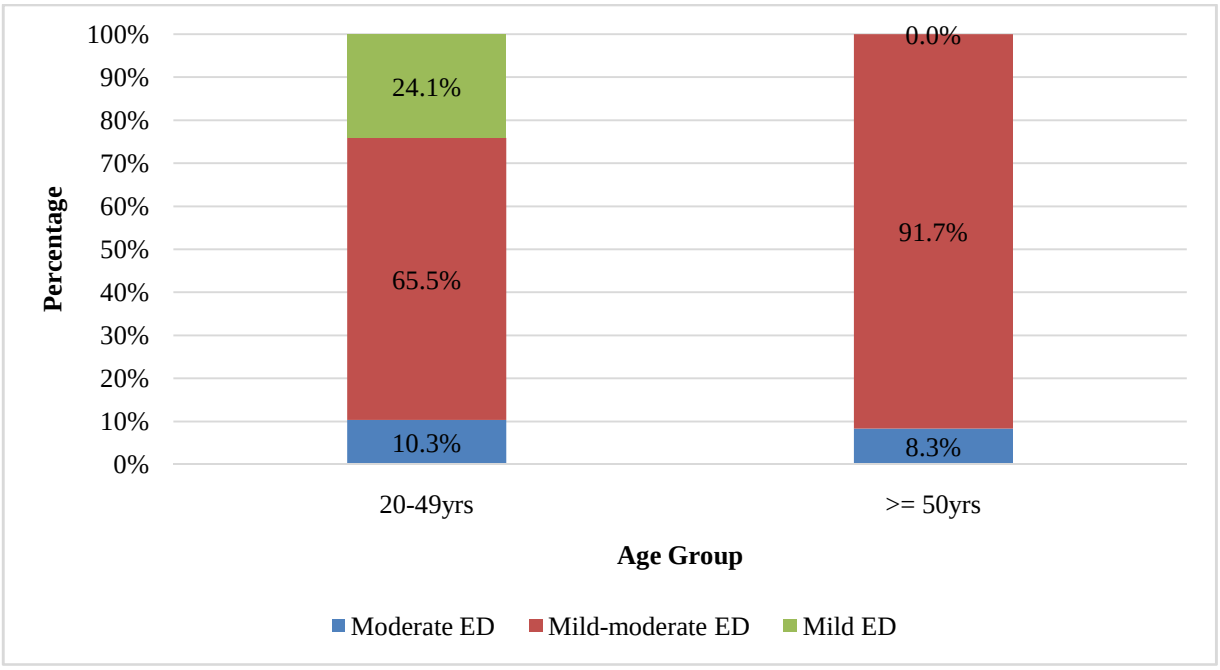


Table 43: Correlation between Erectile Dysfunction and HIV duration in months when serum Testosterone is normal (N=34)

Parameter	Spearman Correlation (rs)	P value
HIV duration in month	-0.456	0.010

There was a weak negative correlation between HIV duration in a month and Erectile dysfunctionwhen serum testosterone was normal in the study population(rs Value: -0.456, P value: 0.010) (Table 43 & Figure 32)

Figure 32: Scatter plot of Erectile Dysfunction and duration in months when serum Testosterone is normal (N=34)

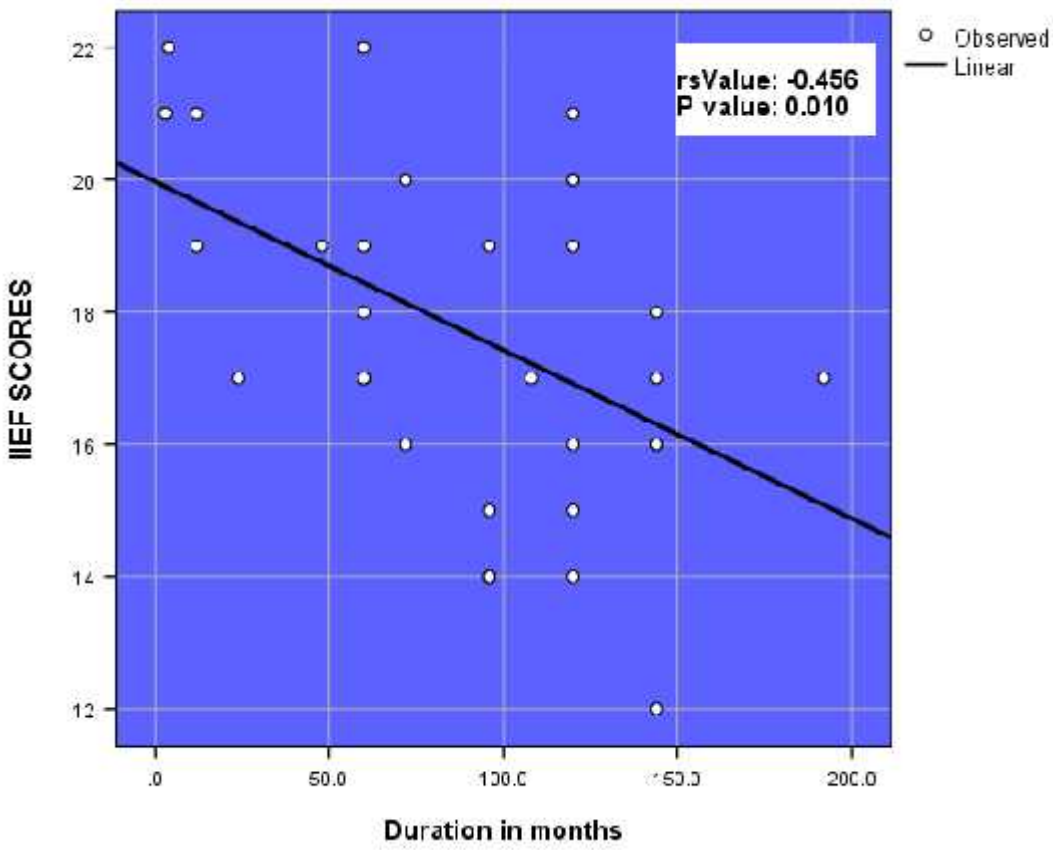


Table 44: Correlation between Erectile Dysfunction and HIV duration in months when serum Testosterone is abnormal (N=41)

Parameter	Spearman Correlation (rs)	P value
HIV duration in month	-0.348	0.028

There was a weak negative correlation between HIV duration in a month and Erectile dysfunctionwhen serum testosterone was abnormal in the study population(rs Value: -0.348, P value: 0.028) (Table 44 & Figure 33)

Figure 33: Scatter plot of Erectile Dysfunction and duration in months when serum Testosterone is abnormal (N=41)

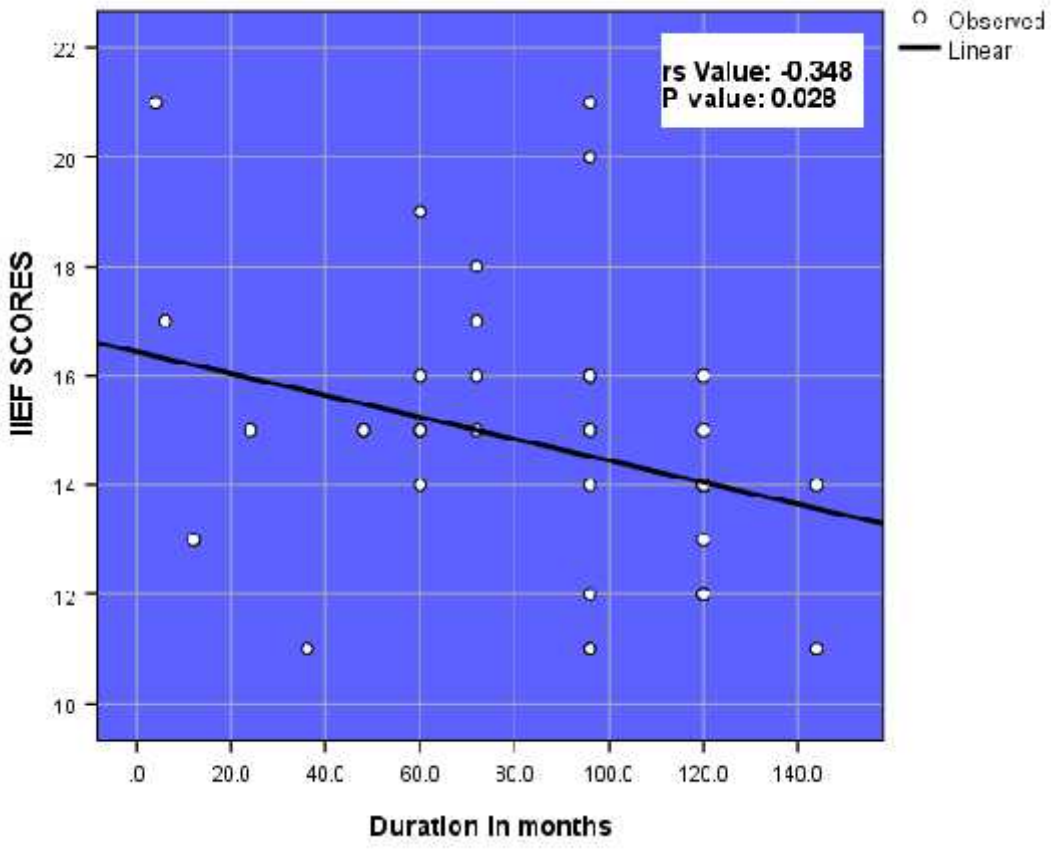


Table 45: Comparison of median IIEF score with CD4 count (N=75)

IIEF score	CD4 Count Median (IQR)	Kruskal Wallis test (P value)
Moderate ED (N=4)	121.50 (43.75 to 240.5)	<0.001
Mild-Moderate ED (N=41)	213 (128 to 342)	
Mild ED (N=27)	451 (268 to 682)	
No ED (N=3)	656 (565 to 854)	

The median CD4 count was 121.50 (IQR 43.75 to 240.5) in moderate ED; it was 213 (IQR 128 to 342) in mild-moderate ED, it was in 451 (IQR 268 to 682) mild ED and 656 (IQR 565 to 854) in No ED. The difference in the median of IIEF score between CD 4 count was statistically significant (P value <0.001). (Table 45)

Table 46: Comparison of median IIEF score with CD4 count (N=75)

IIEF score	CD4 Count Median (IQR)	Mann Whitney U test (P value)
Yes (N=72)	656 (565 to 854)	0.021
No (N=3)	268 (156 to 565)	

The median CD4 counts were 656 (IQR 565 to 854) in IIEF score, and it was 268 (IQR156 to 565) in No. The difference in the median of IIEF score between CD4 count was statistically significant (P value 0.021). (Table 46)

Table 47: Comparison of median IIEF score with CD4 count when serum Testosterone is normal (N=34)

IIEF score	CD4 Count Median (IQR)	Kruskal Wallis test (P value)
Moderate ED (N=0)	0 (0,0)	0.207
Mild-Moderate ED (N=11)	468 (213, 624)	
Mild ED (N=20)	565 (350, 715.8)	
No ED (N=3)	656 (565, 854)	

The median CD4 count in normal serum testosterone was 0 (IQR 0 to 0) in moderate ED; it was 468 (IQR 213, 624) in mild-moderate ED, it was in 565 (IQR 350, 715.8) mild ED and 656 (IQR 565, 854) in No ED. The difference in the median of IIEF score between CD4 count was statistically not significant (P value 0.207). (Table 47)

Table 48: Comparison of median IIEF score with CD4 count when serum Testosterone is abnormal (N=41)

IIEF score	CD4 Count Median (IQR)	Kruskal Wallis test (P value)
Moderate ED (N=4)	121.5 (43.75, 240.5)	0.018
Mild-Moderate ED (N=30)	173 (128, 264.8)	
Mild ED (N=7)	282 (246, 358)	
No ED (N=0)	0 (0,0)	

The median CD4 count in abnormal serum testosterone 121.5 (IQR 43.75, 240.5) in moderate ED, it was 173 (IQR 128, 264.8) in mild-moderate ED, it was in 282 (IQR 246, 358) mild ED and 656 (IQR 565, 854) in No ED. The difference in the median of IIEF score between CD +4 count was statistically not significant (P value 0.207). (Table 48)

Table 49: Comparison of median IIEF score with CD4 count when serumTestosterone is normal (N=34)

IIEF score	CD4 Count Median (IQR)	Mann Whitney U test (P value)
Yes (N=31)	564 (268 to 676)	0.192
No (N=3)	656 (565 to 854)	

The median CD4 counts normal serum testosterone was 564 (IQR 268 to 676) in IIEF score, and it was 656 (IQR565 to 854) in No. The difference it the median of IIEF score between CD4 count was statistically not significant (P value 0.192). (Table 49)

Table 50: Comparison of median IIEF score with CD4 count when serum Testosterone is abnormal (N=41)

IIEF score	CD4 Count Median (IQR)	Mann Whitney U test (P value)
Yes (N=41)	213 (128 to276)	*
No (N=0)	0 (0,0)	

The median CD4 counts abnormal serum testosterone was 213 (IQR 128 to276) in IIEF score. (Table 50)

DISCUSSION

Sexual dysfunction comprehends erectile dysfunction (ED), difficulty in attaining orgasm and loss of libido. HIV infected men have shown the presence of either of one or all these three difficulties. However, erectile dysfunction is most investigated in researches. The incidence of erectile dysfunction among HIV men has been rising, effecting the quality of life among them.[4] Hence, identifying ED among HIV men could help in better management of ED, thus improving their quality of life.

The present study we analyzed 75 HIV infected subjects. The mean age of the study population was 46.12 ± 8.49 years, the minimum age was 21 years, and the maximum age was 60 years in the study population (95% CI 44.17 to 48.07). A similar study by Gomes AR et al[69], had a study population of 245 with a mean age of 48 years (IQR- 15years), another study by Fumaz et al[4],found the median age of 42 years (35,45) similar to our study. The mean BMI was 23.2 ± 2.58 in the study population (95% CI 22.61 to 23.79)

The prevalence of erectile dysfunction in the present study was 96%. The majority (54.67%) of them had mild to moderate ED, followed by 36% with mild ED and 4% of them had no ED. In the age group 20-49yrs, 3 (6%) of the participants had Moderate ED, 23 (46%) of the participants had Mild-Moderate ED, 21 (42%) of the participants had Mild ED, and 3 (6%) of the participants had No ED. In the age group >= 50yrs, 1 (4%) of the participants had Moderate ED, 18 (72%) of the participants had Mild-Moderate ED, 6 (24%) of the participants had Mild ED. A study by Shindel AW, et al[78], found greater ED in men who were older than 40 years and less in younger men. In our study also, we found ED in an older population, but younger HIV men too suffered ED. ED in old age generally could be explained by the fact of

increased comorbid conditions contributing to the prevalence of ED.[80] Increased prevalence of ED in our population was found despite excluding comorbid patients in the study. Hence, HIV patients, even without comorbid conditions, could have other factors which play important role in ED.

A study by Gomez et al[68], found 21.6% of prevalence of ED among their study population (n=134) where 86.2% among them were shown to have severe ED. In a study done by Fumaz et al[4], the prevalence of ED was 58.5%(n=293/521) were the majority(51.5%) of them had mild ED, followed by 33.4% with mild to moderate ED and 10.5% with moderate and 4.4% with severe ED. The high prevalence in the present study compared to other studies could be due to small sample size as compare to other study sample size.

Table 51:Comparing the prevalence of ED among the study population across studies to present study:

Study	Age (mean/ SD/ median) in years	Sample size (n=)	Prevalence %
GomesT et al.[68] 2019	44.8 ± 10.9	134	21.6%
Fumaz et al.[4] 2017	42 years (35,45)	521	58.5%
Present study	46.12 ± 8.49 years	75	96%

Clinical factors:

In general risk factors such as diabetes, hepatopathy, dyslipidemia, hypertension, alcohol intake and vascular illness etc. coexisting with HIV infection have shown to lessen sexual function in men but these factors excluded from our study.[81] Among these conditions, peripheral neuropathy a complication of high viral load and ART therapy has found to be associated with delayed ejaculation.[55] In the present study also we found participants with hepatomegaly, fatty liver but were least

in frequency. But found hypertension on calcium channel blockers which do not cause ED in 14.67% of the study population. A similar prevalence of hypertension (13.4%) among HIV positive men was found in a Brazilian study.[68] In a study by Gomes TV et al[68], among the HIV positive patients (13.4%) presenting with hypertension, 15.2% had erectile dysfunction but found more psychological reasons and low socioeconomic status to cause ED. In our study, we found the majority (63.64%) of hypertensive HIV patients with mild to moderate ED.

A large cohort study (n=1340) study by Hart et al[82], showed ED prevalence of 21% and had included subjects with comorbidities. Even though, this study population had a high proportion of comorbidities (diabetes in 17% and arterial hypertension in 69%) the prevalence of ED was less comparatively and ED prevalence was related to the comorbidities. However, in our study, we excluded subjects with comorbidities, but still found a prevalence of hypertension and the presence of ED among this study population. Thus, we can conclude HIV itself to be one of the reasons for ED among our study group with ED.

In addition to the overall health, anxiety and depression along with fear of transmission of HIV and with HIV by itself is accountable for ED in HIV positive patients.[83] In our study, we found 32% of them having mild depression, followed by 34.67% with Minimal depression and 33.33% with Moderate depression.

The mean Haemoglobin was 11.31 ± 2.49 in the study population, minimum haemoglobin was 7.40, and maximum haemoglobin was 17.50 in the study population (95% CI 10.74 to 11.89). Similar mean haemoglobin (11.2±2.8) was found in a study by Aggarwal J et al.[84]Among the study population in the Anemia patients, 4 (10%) of the participants had Moderate ED, 25 (62.5%) of the participants had Mild-Moderate ED, 10 (25%) of the participants had Mild ED and 1 (2.5%) of the participants had no

ED. We found a significant frequency of HIV patients with Anemia affected by ED. It is explained that hypogonadism results due to low levels of free testosterone (<300nh/dl) from early morning samples which are accompanied with other clinical features such as sexual dysfunction, muscle mass and weight loss, tiredness, depression and anemia.[10] there is the least literature on the association of anemia by itself as a factor for ED in HIV men. Hence the present study results could be further researched with larger sample size and inclusion of comorbid HIV patients. In addition, we found no statistically difference between haemoglobin and IIEF score groupwhen serum testosterone was normal in the study population for the P value of 0.994.so we can conclude though anemia is not statistically significant but clinically significant in our study.

In our study, a weak positive correlation between BMI and Erectile dysfunctionwas foundwhen serum testosterone was abnormal in the study population(rs Value: 0.097, P value: 0.546). In contrast, a study by Crum-Cianflone, N et al[53], found increasing age and greater BMI to be positively associated with hypogonadism. Usually, HIV positive patients experience weight loss with muscle loss and low levels of testosterone.[10] In a systematic review it was found that testosterone replacement therapy improved muscle mass and body mass in the HIV positive men.[85]

In a study by Shindel AW, et al[78], the mean IIEF scores with standard deviation were 25.3±6.1 in a cohort of HIV positive men. In our study, we found mean IIEF Scores to be 16.15 ± 2.93 (95% CI 15.47 to 16.82). Among the study population, IIEF score was 16 (IQR 14.25 to 18.75) for Mild depression, 18 (IQR 16.75 to 20) for Minimal depression and 14 (IQR 12.50 to 15) for moderate depression severity. The relation between depression severity and IIEF score was

statistically significant (P value <0.001). In Fumaz et al[4], study found (according to the IIEF-5), the occurrence of all degrees of ED was 58.5% while among them, mild ED was in 51.5%, mild to moderate in 33.4%, moderate in 10.5% and severe in 4.4%.

Among the study population in Q1, 34.67% gave IIEF score of 3, 53.33% gave IIEF score of 4, 12% gave IIEF score of 5. In Q2, 12% gave IIEF score of 2, 52% gave IIEF score of 3, 34.67% gave IIEF score of 4, 1.33% gave IIEF score of 5. In Q3, 22 (29.33%) gave IIEF score of 2, 38 (50.67%) gave IIEF score of 3, 15 (20%) gave IIEF score of 4. In Q4, 14 (18.67%) gave IIEF score of 2, 35 (46.67%) gave IIEF score of 3, 24 (32%) gave IIEF score of 4, 2 (2.67%) gave IIEF score of 5. In Q5, 10 (13.33%) gave IIEF score of 2, 45 (60%) gave IIEF score of 3, 19 (25.33%) gave IIEF score of 4, 1 (1.33%) gave IIEF score of 5. In a study by Santi D et al[86], the IIEF-15, the ED was weakened in 60.3% of patients, and a 13.2% had severe ED. Among these, 11% of participants had been on PDE5-inhibitors where these patients showed higher scores at questionnaires. However, in our study, we did not analyze the correlation type of drug protocol to IIEF score.

Our study population, IIEF score was 16 (IQR 14.25 to 18.75) for Mild depression, 18 (IQR 16.75 to 20) for Minimal depression and 14 (IQR 12.50 to 15) for moderate depression severity($p<0.001$). Among the study population in the mild depression severity when serum testosterone was abnormal, 9 (75%) of the participants had Mild-Moderate ED, 3 (25%) of the participants had Mild ED. In the minimal depression severity, 1 (16.67%) of the participants had Moderate ED, 2 (33.33%) of the participants had Mild-Moderate ED, 3 (50%) of the participants had Mild ED. In the moderate depression severity, 3 (13.04%) of the participants had Moderate ED, 19 (82.61%) of the participants had Mild-Moderate ED and 1 (4.35%)

of the participants had Mild ED. Numerous studies have related depression to ED, delayed ejaculation and no desire for sex.[87,88] A study by de Ryck et al[87], found sexual function in the majority of HIV positive men due to depression.

Correlation of ART, testosterone and ED:

The reasons for the decline in sexual dysfunction in HIV infected men could be attributed to antiretroviral drugs. However, the role of Antiretroviral drugs in sexual dysfunction is still a debate. Few studies[5,89], have shown antiretroviral drugs to cause sexual dysfunction, but some have shown no association.[53,90] However, our study participants on retroviral therapy showed ED. Among the study population in ART regimen, 53 (70.67%) of the participants had TLE, 10 (13.33%) of the participants had ZLE.

Among the study population in the TLE ART regimen when serum testosterone was normal, 9 (36%) of the participants had Mild-Moderate ED, 15 (60%) of the participants had Mild ED and 1 (4%) of the participants had no ED. In the ZLE ARTregimen, 1 (33.33%) of the participants had Mild-Moderate ED, 2 (66.67%) of the participants had Mild ED. In a study by Aggarwal et al[84], found Mean free testosterone and FSH to be significantly greater in subjects on ART than in those where not on ART ($P = 0.028$ and $P = 0.045$, respectively), but did could relate to the exact ART drug or their combination drug to have a significant correlation with levels of testosterone. Hence it is suggested that ART therapy does improve the testosterone levels in which the HIV patients are deficient off.

Among the study population in the TLE ART regimen when serum testosterone was abnormal, 3 (10.71%) of the participants had Moderate ED, 21 (75%) of the participants had Mild- Moderate ED and 4 (14.29%) of the participants

had Mild ED. In the ZLE ART regimen, 1 (14.29%) of the participants had Moderate ED, 5 (71.43%) of the participants had Mild-Moderate ED, 1 (14.29%) of the participants had Mild ED.

Among the study population in the TLE ART regimen, 3 (5.66%) of the participants had Moderate ED, 30 (56.6%) of the participants had Mild-Moderate ED, 19 (35.85%) of the participants had Mild ED, and 1 (1.89%) of the participants had no ED. In the ZLE ART regimen, 1 (10%) of the participants had Moderate ED, 6 (60%) of the participants had Mild-Moderate ED and 3 (30%) of the participants had Mild ED. The frequency of HIV patients not on ART were 16%(n=12) and found mild to moderate and moderate ED in 41.67% each and no ED in 16.67%. In a study by Lamba et al[91], it was found that HIV patients with HAART had found ED in 25% whereas, in HIV men who were not on HAART was found ED in 26%. Our study found the prevalence of ED in 8.3% of HIV patients, not on ART therapy.

Almost majority of antiretroviral drugs have shown to be related to various degree of Sexual impairment. Where indinavir is the related top highest frequency of dysfunction with nevirapine related to lowest, hence if one identifies the principle drug which leads to sexual impairment, it should be replaced with nevirapine or atazanavir.[6]

The mean ART Regimen Duration was 4.76 ± 2.31 in the study population, minimum duration was 1, and the maximum duration was 12 in the study population (95% CI 4.18 to 5.34).In a study by Pongener N, et al[74], found no significant correlation between the duration of ART of the patients and gonadal dysfunctions. Similarly, in the present study we found a weak negative correlation between ART regimen duration and Erectile dysfunctionwhen serum testosterone was abnormal or normal in the study population(rs Value: -0.288, P value: 0.093). Our study findings

were in agreement with Jain et al[92], and Rietschel, Pet al.[93] However, this finding has to be further studied as we did not compare the levels of testosterone prior to the start and on treatment. Low sexual desire and deficient testosterone levels in HIV subjects could be associated with the advancement of HIV illness or could be attributed to morbid conditions or with mental status.

Comparison of serum testosterone with ED:

Patients with HIV infection exhibit a wide range of endocrine abnormalities; one of them includes testosterone deficiency leading to erectile abnormalities.[74] In the past studies, the prevalence of low testosterone in HIV patients was known to be ranging from 29-50%.[94,95] But after the proposal of novel active retroviral drugs (HAART), the researchers have found lesser prevalence ranging from 9-16%.[73,70]In our study, the mean Serum Testosterone was 8.62 ± 6.99 in the study population, minimum Serum Testosterone was 0.09, and maximum Serum Testosterone was 28.30 (95% CI 7.01 to 10.23). In a study byPongener N et al[74], found the mean CD4 count to be 442.63 ± 276.97 cells/mm^3 and mean testosterone level to be 432.73 ± 207.169 ng/dL.

In the present study population, in the normal serum testosterone individuals (45.3%), 32.35% of the participants had Mild-Moderate ED, 58.82% of the participants had Mild ED, and 8.82% of the participants had no ED. In the abnormal serum testosterone (54.6%), 9.76% of the participants had Moderate ED, 73.17% of the participants had Mild-Moderate ED, and 17.07% of the participants had Mild ED. In our study, we found that nearly half of the patients had testosterone deficiency. In a large cohort study(n=1325) by Rochira V et al[73], found total serum Testosterone levels less than 300 ng/dL (10.4 nmol/L) in 16% of their study subjects, with the

maximum rate of total low Testosterone in men aged between 40 and 60 years. This study also found a significant low testosterone levels in younger men (10.6%) aged 30-39 years. However, such correlation was not made in our study, but we found significant low levels of testosterone in the study population, and we assessed free testosterone levels which is more accurate. Indian studies[77,13], have shown the varied prevalence of low testosterone hormone (13.3% to 33%). In a study by Dutta D et al[75], found nearly 80.68% of them with low testosterone levels in 39.11% of the study population with hypogonadism. Hypogonadism was significant in older males with longer duration of HIV infection.[75] In our study also we found the mean duration in Months was 94.67 ± 36.01, minimum duration was 12, and the maximum duration was 192 in the study population (95% CI 85.60 to 103.73). Our study also found a positive correlation between Serum testosterone and Erectile disfunctionin the study population (rs Value: 0.680, P value: <0.001).

A large cohort study which included 1776 HIV infected men observed a correlation between free testosterone, bioavailable testosterone and total testosterone with ED.[96] Several studies have showed total testosterone(TT) with no correlation to the risk of ED[97,98], and severity, but low TT in older individuals had related to ED.[99] Few studies[100,101], have shown FT and not TT to be correlated to ED when 5 domain of IIEF were examined. The present study found a positive correlation between Serum testosterone and Erectile disfunctionin the study population (rs Value: 0.680, P value: <0.001) Our study estimated FT and found low FT correlated to ED which was in agreement with a study Huang Y et al[102], were low FT among young HIV individuals correlated with ED.

Correlation of CD4 count and testosterone to ED.

From previously mentioned study Dutta et al[75], its was found older-aged males and low CD4 count (baseline) and vit D deficiency to be the predictors for hypogonadism in HIV men. However, it has been contrasting results in relation to CD4 count and hypogonadism.[103,60] In our study, we found the mean CD4 Count to be 349.75 ±240.52, minimum CD4 Count was 17, and maximum CD4 Count was 854 (95% CI 294.41 to 405.08). In a study by Aggarwal et al[84], the range of CD4 count was 33.00-841.00 and mean SD was 250.16±20.53, whereas, in our study, the mean CD4 scores in the study population were high inferring good immune response.

There was a weak positive correlation between CD4 count and Erectile dysfunctionwhen serum testosterone was normal and abnormal in the age group 20-49yrs(rs Value: 0.316, P value: 0.163,rs Value: 0.482, P value: 0.008 respectively). But in age group greater than 50 years (normal and abnormal T levels) of age showed a weak correlation between CD4 count and testosterone level. Hence, our study showed no correlation of CD4 count to testosterone levels with across different age groups.

The median CD4 count was 121.50 (IQR 43.75 to 240.5) in moderate ED, it was 213 (IQR 128 to 342) in mild-moderate ED, it was in 451 (IQR 268 to 682) mild ED and 656 (IQR 565 to 854) in No ED. The difference in the median of IIEF score between CD +4 count was statistically significant (P value <0.001). In a study by Aggarwarl et al[84], found an association of low testosterone levels in patients with immunodeficiency. Although such association was not analyzed in our study, we did find low CD4 count with abnormal testosterone in the subjects with mild-moderate ED. But the difference in the median of IIEF score between CD4 count was

statistically not significant (P value 0.207). In our study, we found median CD4 count was 656 (IQR 565 to 854) in IIEF score, and it was 268 (IQR156 to 565) in No. The difference in the median of IIEF score between CD4 count was statistically significant (P value 0.021). The study by Aggarwal et al[84], as mentioned previously, have found a significant relation between the prevalence of hypogonadism and level of CD4 counts as the frequency of hypogonadism increased with a decrease in CD4 counts. Further, in Meena et al[77], study they found a significant correlation between free testosterone and CD4 counts. However, in our study, we found the median CD + 4 counts abnormal serum testosterone was 213 (IQR 128 to276) in IIEF score.

In our study the median CD4 count in normal serum testosterone was 0 (IQR 0 to 0) in moderate ED, it was 468 (IQR 213, 624) in mild-moderate ED, it was in 565 (IQR 350, 715.8) mild ED and 656 (IQR 565, 854) in No ED. The difference in the median of IIEF score between CD4 count was statistically not significant (P value 0.207). The median CD4 count in abnormal serum testosterone 121.5 (IQR 43.75, 240.5) in moderate ED, it was 173 (IQR 128, 264.8) in mild-moderate ED, it was in 282 (IQR 246, 358) mild ED and 656 (IQR 565, 854) in No ED. The difference in the median of IIEF score between CD4 count was statistically not significant (P value 0.207).

The median CD4 counts normal serum testosterone was 564 (IQR 268 to 676) in IIEF score, and it was 656 (IQR565 to 854) in No. The difference it the median of IIEF score between CD4 count was statistically not significant (P value 0.192). Hence in our study, we found an insignificant association of CD4 count with testosterone and ED as the CD4 count was found to be high with good immune response and no other comorbidities in subjects.

LIMITATION&RECOMMENDATION:

- The present study results cannot be generalized due to the relatively small sample size and design of the study.
- Further research with large sample size and longitudinal study allows the researcher to appropriately correlate with HAART treatment, comorbidities, levels of testosterone and erectile dysfunction in HIV men.
- IIEF scoring has a validated protocol for men who have sex with men (MSM) and heterosexual group; however, our study did not analyze or group the study population
- Although we found anemia to be associated with ED, the type of Anemia was not elicited, and hence future research on the association of anemia in HIV men with ED is required.

STRENGTH OF STUDY:

1. We have compared free testosterone with erectile dysfunction and other clinical factors which is more sensitive and specific.

2. We have taken larger sample size as compare to other similar studies.

3. We have excluded comorbid condition in HIV patient so in our study we concluded that HIV per se associated with erectile dysfunction.

CONCLUSION

1. In the present study we analyzed 75 HIV infected subjects with a mean age of 46.12 ± 8.49 years.
2. The prevalence of erectile dysfunction was 96%. The majority (54.67%) of them had mild to moderate ED. HIV individuals aged more than 50 years had a greater prevalence of mild to moderate ED (N=18,72%) as compared to younger age individuals with 20-49 years of age (N=23,46%).
3. Erectile dysfunction was seen in patients with abnormal (low) testosterone and also with normal testosterone.Despite the prevalence of erectile dysfunction in both study groups, number of HIV patients with erectile dysfunction and low testosterone was more as compare to patients with erectile dysfunction with normal testosterone(rs Value: 0.680, P value: <0.001).
4. In my study, most of patients who had Erectile dysfunction with normal testosterone had minimal depression (n=20) as compared to most of the patients who had erectile dysfunction with low testosterone had moderate depression (n=23) (P value <0.001).
5. Anemia is found in almost half(N-41) of patients with erectile dysfunction and low testosterone which is clinically significant but statistically not significant.
6. There was no association of Age, ART and CD4 count with Free Testosterone levels (normal or abnormal) in patients with ED among HIV men.

SUMMARY

Sexual life is one of the important needs of having a healthy living with a good quality of life. HIV infection, especially in men, has shown to have low sexual activity due to various physiological and psychological reasons. From literature, it is learnt that there exists a high prevalence of erectile dysfunction in HIV positive men. Many studies have correlated testosterone hormone to various degrees of ED, but have found contrary results. Likewise, many clinical factors in HIV men have been found in association with ED. Hence the present study aimed to find the prevalence of ED and correlate clinical factors and testosterone hormone to ED in HIV positive patients.

Our study analyzed 75 HIV infected subjects. The mean age was 46.12 ± 8.49 years, the minimum age was 21 years, and the maximum age was 60 years (95% CI 44.17 to 48.07). The prevalence of erectile dysfunction in the present study was 96%. The majority (54.67%) of them had mild to moderate ED, followed by 36% with mild ED, and 4% of them had no ED. In the age group 20-49yrs, 3 (6%) of the participants had Moderate ED, 23 (46%) of the participants had Mild-Moderate ED, 21 (42%) of the participants had Mild ED, and 3 (6%) of the participants had No ED. In the age group >= 50yrs, 1 (4%) of the participants had Moderate ED, 18 (72%) of the participants had Mild-Moderate ED, 6 (24%) of the participants had Mild ED. Hypertension was found in 14.67% of the study population. The mean IIEF Scores to be 16.15 ± 2.93 (95% CI 15.47 to 16.82). The relation between depression severity and IIEF score was statistically significant (P value <0.001).

The mean ART Regimen Duration was 4.76 ± 2.31 in the study population, minimum duration was 1, and the maximum duration was 12 (95% CI 4.18 to 5.34). Among the study population in the TLE ART regimen when serum testosterone was normal, 9 (36%) of the participants had Mild-Moderate ED, 15 (60%) of the participants had Mild ED and 1 (4%) of the participants had no ED. In the ZLE ART regimen, 1 (33.33%) of the participants had Mild-Moderate ED, 2 (66.67%) of the participants had Mild ED.

There was a weak positive correlation between CD 4+ count and Erectile dysfunction when serum testosterone was normal in the age group 20-49yrs (rs Value: 0.316, P value: 0.163). There was a moderate positive correlation between Serum testosterone and Erectile dysfunction in the study population (rs Value: 0.680, P value: <0.001) There was a negative correlation between ART regimen duration and Erectile dysfunction when serum testosterone was abnormal in the study population (rs Value: -0.288, P value: 0.093) There was a negative correlation between Age and Erectile dysfunction when serum testosterone was normal in the study population (rs Value: -0.568, P value: <0.001) There was a negative correlation between Age and Erectile dysfunction when serum testosterone was abnormal in the study population (rs Value: -0.459, P value: 0.003) There was a weak positive correlation between BMI and Erectile dysfunction when serum testosterone was abnormal in the study population (rs Value: 0.097, P value: 0.546). Hence this study results showed that ED prevalent in HIV patients with depression and patient having low testosterone. There was a positive correlation between testosterone hormone and ED in HIV positive patients.

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ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to-be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
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Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/52 **Date: 24/11/2018**

To,
REG. NO.BG0118008
PG student in Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"TO STUDY CLINICAL FACTORS AND SERUM TESTOSTERONE IN RELATION TO ERECTILE DYSFUNCTION IN HIV INFECTED MEN AT KLE'S DR. PRABHAKAR KORE HOSPITAL & MRC, BELAGAVI – A ONE YEAR OBSERVATIONAL CROSS SECTIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE II

CONSENT FORM

Dear Mr./Mrs./Dr. _____, you are kindly requested to enroll yourself in a research study tilted "TO STUDY CLINICAL FACTORS AND SERUM TESTOSTERONE IN RELATION TO ERECTILE DYSFUNCTION IN HIV INFECTED MEN AT KLE'S DR PRABHAKAR KORE HOSPITAL & MRC,BELAGAVI-A ONE YEAR OBSEVATIONAL CROSS SECTIONAL STUDY" being conducted by , a post graduate student in M.D.GENERAL MEDICINE and the study will be carried out under the direct supervision and guidance of , Professor, Department of Medicine, Jawaharlal Nehru Medical College, Belagavi.

You have been requested to participate in this as you fit into the laid out criteria for a study 'subject'/ participant.

Your participation in study is voluntary. During the study you will be undergoing an interview session. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY: "TO STUDY CLINICAL FACTORS AND SERUM TESTOSTERONE IN RELATION TO ERECTILE DYSFUNCTION IN HIV INFECTED MEN AT KLE'S DR PRABHAKAR KORE HOSPITAL & MRC,BELAGAVI-A ONE YEAR OBSEVATIONAL CROSS SECTIONAL STUDY"

"PURPOSE OF THE STUDY:

1. To determine the correlation between erectile dysfunction with clinical factors and serum testosterone. .

2. To determine the prevalence of erectile dysfunction among hiv infected men

PROCEDURES INVOLVED:

If you agree to enroll yourself in my study, you will be subjected to semi-structured questionnaires.

,

With the help of a IIEF-5 QUESTIONAIRE, The patient's severity is assessed and on the basis of score classified into severe ED,Moderate ED,Mild ED,no ED.The study requires following investigations:1.CBC 2.LFT 3.RFT 4.LIPID PROFILE.

5.CD4 COUNT 6.SERUM FREE TESTOSTERONE.6.CXR 7.USG ABDOMEN .

RISKS AND BENEFITS:

There are no potential risks involved in this study.

Benefits of taking part in this research:

The investigator does not promise or guarantee that you will receive any benefit in the study however it will be better understanding of the factors affecting erectile dysfunction so that effective treatment or other intervention can be given to improve the quality of life

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES:

Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY:

All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your

informed & written consent.

The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except:

- In emergency to protect your rights AND welfare.
- If required by law.

AUTHORIZATION TO PUBLISH RESULT:

The results of the study may be used to publish an article. When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No additional costs shall be incurred upon you for the purpose of this study. It is purely being done with the idea of research and all the cost of study will be borne by the investigator. There will be not be any remuneration, reimbursement, compensation or free medical care.

QUESTIONS/CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

In case of the queries during study or in future you may contact following persons,

1. DR.Roopa M Bellad,
Chairman,
J.N.M.C Ethical Committee
for Human Research

Proffesor
Department Of General
Medicine
JNMC, Belagavi

Investigator,
PG in General Medicine,
JNMC, Belagavi.

STATEMENT OF CONSENT

I/my relative have/has read and have/has completely understood the entire information given in the consent form, which explains all the details of the study, i.e, the purpose, procedure involved, risks & benefits, privacy & confidentiality, incentives and the authorization to publish the results of the study. My/my relative's signature in the space provided for signature below indicates that I/my relative have/has voluntarily agreed to participate in the study. I/my relative may withdraw my/my relative's participation for any reason or may be withdrawn by the investigator from the study for any reason at any time. I/my relative am/is not giving up any of my/my relative's legal rights by signing this consent form.

Signature of the participant with date: _____

Name of the participant: _____

Signature of the authorized representative with date: _____

Name of the authorized representative: _____

Relationship of authorized person: _____

Signature of the witness with date: _____

Name of the witness: _____

Signature of the Investigator with date: _____

ANNEXURE III

PROFORMA

Patient Data

Name -: IP/OP :-

Age / Sex :- Contact Number :-

Address :-

General Physical Examination :-

- Pulse :- / min. : RR :- cycles / min.
- BP:- / mm Hg. ; Temperature :- ^{o}F

- WEIGHT:- Kgs . HEIGHT :- = cm.

- BMI:- : Waist Circumference :- cm .

Past History:-

SYSEMIC EXAMINATION:-

- CVS :-

- R/S:-
- CNS:-
- P/A:-
- Genitals examination:-

DIAGNOSIS:-

QUESTIONNAIRE

NAME:
AGE:
QUALIFICATION:

GENDER:
MARRIED:
DESIGNATION:

1. Do you have knowledge about HIV disease ?

Ans:

2. Did you ever hospitalised after Hiv infection ?

Ans

3. Are you on any medication for HIV or any other disease like Tuberculosis,Diabetes,Hypertension and any other infections? Which?
   Dose? Whether taking regularly?

Ans:

4. Do you have Hypertension? Duration? Medications?

Ans:

5. Do you have Diabetes Mellitus? Duration? Medications?

Ans:

6. Do you suffer from any other disease? If yes,Rx?

Ans:

7. Do you suffer from stress because of work?

Ans:

8. Do you have any habits?

Ans:

9. Have you lost or gained weight?

Ans:

10.Are you Taking ART?

Ans:

The IIEF-5 Questionnaire (SHIM)

The IIEF-5 Questionnaire (SHIM)

Please encircle the response that best describes you for the following five questions:

Over the past 6 months:					
1. How do you rate your confidence that you could get and keep an erection?	Very low 1	Low 2	Moderate 3	High 4	Very high 5
2. When you had erections with sexual stimulation, how often were your erections hard enough for penetration?	Almost never or never 1	A few times (much less than half the time) 2	Sometimes (about half the time) 3	Most times (much more than half the time) 4	Almost always or always 5
3. During sexual intercourse, how often were you able to maintain your erection after you had penetrated your partner?	Almost never of never 1	A few times (much less than half the time) 2	Sometimes (about half the time) 3	Most times (much more than half the time) 4	Almost always or always 5
4. During sexual intercourse, how difficult was it to maintain your erection to completion of intercourse?	Extremely difficult 1	Very difficult 2	Difficult 3	Slightly difficult 4	Not difficult 5
5. When you attempted sexual intercourse, how often was it satisfactory for you?	Almost never or never 1	A few times (much less than half the time) 2	Sometimes (about half the time) 3	Most times (much more than half the time) 4	Almost always or always 5

Total Score: _____

1-7: Severe ED 8-11: Moderate ED 12-16: Mild-moderate ED 17-21: Mild ED 22-25: No ED

PATIENT HEALTH QUESTIONNAIRE (PHQ-9)

NAME:_____ DATE:_____

Over the last 2 *weeks*, how often have you been bothered by any of the following problems?
(use "✓" to indicate your answer)

	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself—or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed. Or the opposite – being so figety or restless that you have been moving around a lot more than usual	0	1	2	3
9. Thoughts that you would be better off dead, or of hurting yourself	0	1	2	3
	add columns	+	+	

(Healthcare professional: For interpretation of TOTAL, please refer to accompanying scoring card). TOTAL:

10. If you checked off *any problems*, how *difficult* have these problems made it for you to do your work, take care of things at home, or get along with other people?	Not difficult at all _____ Somewhat difficult _____ Very difficult _____ Extremely difficult _____

PHQ-9 Patient Depression Questionnaire

For initial diagnosis:

1. Patient completes PHQ-9 Quick Depression Assessment.
2. If there are at least 4 ✓s in the shaded section (including Questions #1 and #2), consider a depressive disorder. Add score to determine severity.

Consider Major Depressive Disorder

- if there are at least 5 ✓s in the shaded section (one of which corresponds to Question #1 or #2)

Consider Other Depressive Disorder

- if there are 2-4 ✓s in the shaded section (one of which corresponds to Question #1 or #2)

Note: Since the questionnaire relies on patient self-report, all responses should be verified by the clinician, and a definitive diagnosis is made on clinical grounds taking into account how well the patient understood the questionnaire, as well as other relevant information from the patient.
Diagnoses of Major Depressive Disorder or Other Depressive Disorder also require impairment of social, occupational, or other important areas of functioning (Question #10) and ruling out normal bereavement, a history of a Manic Episode (Bipolar Disorder), and a physical disorder, medication, or other drug as the biological cause of the depressive symptoms.

To monitor severity over time for newly diagnosed patients or patients in current treatment for depression:

1. Patients may complete questionnaires at baseline and at regular intervals (eg, every 2 weeks) at home and bring them in at their next appointment for scoring or they may complete the questionnaire during each scheduled appointment.
2. Add up ✓s by column. For every ✓: Several days = 1 More than half the days = 2 Nearly every day = 3
3. Add together column scores to get a TOTAL score.
4. Refer to the accompanying **PHQ-9 Scoring Box** to interpret the TOTAL score.
5. Results may be included in patient files to assist you in setting up a treatment goal, determining degree of response, as well as guiding treatment intervention.

Scoring: add up all checked boxes on PHQ-9

For every ✓ Not at all = 0; Several days = 1;
More than half the days = 2; Nearly every day = 3

Interpretation of Total Score

Total Score	Depression Severity
1-4	Minimal depression
5-9	Mild depression
10-14	Moderate depression
15-19	Moderately severe depression
20-27	Severe depression

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ANNEXURE IV

KEY TO THE MASTER CHART

Age Group	1	20-49yrs
	2	>= 50yrs
Gender	1	Male
	2	Female
Depression Severity	1	Mild
	2	Minimal
	3	Moderate
Comorbidities	1	HTN
	2	No
Art regimen	1	TLE
	2	ZLE
	3	NOT ON ART

MASTER SHEET

SERIAL NO.	IP.NO	Age group	Age	Gender	BMI	Diagnosis	Q1	Q2	Q3	Q4	Q5	IIEF Score	PHQ-9	Depression Severity	Serum testosterone	CD 4+ COUNT	Comorbidities	Duration in months	Art regimen	Art regimen duration	Haemoglobin	Platelet	TLC	Creatine	Total bilirubin
1	966743	2	53	1	22.7	ACUTE APPENDICITIS	4	4	4	4	4	MILD ED-20	4	2	13.800	564	2	120	1	4	12.9	2.77	11.10	1.28	1.40
2	967562	2	55	1	23.20	BPH	4	4	4	4	4	MILD ED-20	4	2	18.580	451	2	120	1	6	11.9	1.81	6.50	1.09	0.23
3	934907	1	48	1	20.50	PYLONEPHRITIS	5	4	4	5	4	NO ED-22	2	2	26.020	656	2	60	1	3	10.9	2.86	7.80	1.29	0.30
4	960242	2	52	1	20.50	SYNCOPE	3	2	3	3	3	MILD-MOD ED-14	11	3	0.087	116	1	60	1	3	9.5	2.97	11.20	1.10	0.35
5	931106	1	47	1	25.00	ORAL CANIDIASIS	4	3	2	3	3	MILD-MOD ED-15	6	1	5.010	318	2	24	1	2	10.3	1.97	2.23	0.85	0.30
6	5175885	1	42	1	22.10	ACALCULOUS CHOLECYSTITIS	5	4	4	4	4	MILD ED-21	2	2	20.770	407	2	3	3		11.3	3.43	5.97	0.90	0.50
7	938218	1	49	1	21.60	URINARY TRACT INFECTION	3	2	2	2	3	MODERATE ED-11	12	3	0.624	85	1	36	2	2	9.8	4.08	15.80	0.83	0.80
8	940113	2	50	1	19.20	CATARACT	4	3	3	3	3	MILD-MOD ED-16	4	2	18.560	60	2	120	1	8	14.8	1.80	8.60	0.72	1.00
9	943098	2	52	1	22.00	LEFT LOWER LIMB DVT	3	2	2	2	2	MODERATE ED-11	4	2	2.690	158	2	144	1	5	7.4	3.61	9.33	1.60	1.20
10	1012987	1	30	1	24.20	ACUTE GASTROENTERITIS	5	4	4	4	4	MILD ED-21	1	2	7.950	287	2	4	3		17.0	3.17	9.27	1.50	0.70
11	969186	2	52	1	25.70	BPH	3	3	3	3	3	MILD-MOD ED-15	8	1	10.170	268	1	96	1	5	12.4	2.85	8.50	1.40	0.80
12	1013369	2	54	1	26.60	VIRAL FEVER	3	2	2	3	3	MODERATE ED-13	13	3	0.428	166	2	120	1	5	8.6	1.50	5.10	1.10	0.20
13	970855	1	40	1	24.61	DENGUE FEVER	5	4	3	3	4	MILD ED-19	4	1	6.470	854	2	60	2	2	14.2	3.38	7.50	0.69	0.30
14	894101	2	53	1	22.50	ACUTE PANCREATITIS	4	3	3	4	3	MILD ED-17	3	1	8.830	264	2	144	2	7	9.4	1.37	4.60	0.77	1.50
15	986156	1	40	1	24.30	TIBIA BONE FRACTURE	3	3	3	3	3	MILD-MOD ED-15	13	3	1.120	128	2	60	1	5	7.9	1.50	9.60	0.90	0.30
16	1013844	2	52	1	24.00	COPD	4	3	2	2	2	MODERATE ED-13	12	3	1.210	356	2	12	3		11.0	52.00	13.20	1.26	1.31
17	1012031	2	60	1	23.00	CVA	3	3	3	3	3	MILD-MOD ED-15	6	2	4.960	50	1	48	3		8.0	94.00	5.00	1.20	0.19
18	981229	1	41	1	21.50	CHOLELITHISIS	4	4	4	4	4	MILD ED-20	8	2	7.410	246	2	96	1	5	8.6	1.50	5.50	1.20	1.10
19	962907	1	36	1	22.20	ACUTE GASTROENTERITIS	5	4	4	4	4	MILD ED-21	4	1	10.270	77	2	12	1	1	11.2	1.47	3.90	1.40	0.42
20	5604400	1	46	1	24.33	CONSTIPATION	4	4	3	4	4	MILD ED-19	4	1	14.270	549	2	60	1	3	12.4	3.50	8.20	1.10	0.80
21	5537760	2	50	1	23.70	ACUTE PANCREATITIS	4	3	3	3	3	MILD-MOD ED-16	9	1	4.370	136	2	96	2	5	9.9	2.20	5.90	1.20	0.63

22	5551190	1	43	1	19.10	BRONCHIAL ASTHAMA	4	3	3	2	3	MILD-MOD ED-15	10	3	3.030	263	2	60	1	2	8.8	1.05	3.70	0.80	0.40
23	954188	1	21	1	24.30	DENGUE FEVER	4	3	3	3	3	MILD-MOD ED-16	9	1	4.080	347	2		3		11.8	2.90	13.20	1.20	0.60
24	958485	1	44	1	21.60	URINARY TRACT INFECTION	4	3	3	3	3	MILD-MOD ED-16	12	3	4.910	528	2	72	1	2	11.7	3.25	4.90	0.80	0.53
25	959193	1	42	1	22.20	VIRAL FEVER	4	4	3	4	3	MILD ED-18	4	2	9.190	356	2		3		9.0	1.50	8.60	1.30	0.29
26	995569	1	45	1	24.30	PYLONEPHRITIS	4	3	3	3	3	MILD-MOD ED-16	12	3	1.880	128	2	60	1	2	10.0	3.27	11.00	1.50	1.10
27	5551478	2	60	1	19.20	DYSPESIA	3	3	2	2	2	MILD-MOD ED-12	4	1	10.740	42	2	144	1	8	14.7	1.54	8.50	1.17	0.90
28	998748	2	58	1	20.90	MENINGITIS	3	2	2	3	2	MILD-MOD ED-12	14	3	0.512	17	2	120	1	8	10.2	1.94	4.90	0.81	0.33
29	997026	1	48	1	24.30	SEVERE OESOPHAGITIS	4	4	3	3	3	MILD ED-17	8	2	15.390	33	1	60	2	5	11.6	1.93	1.90	0.78	0.26
30	995506	2	60	1	25.90	AKI SEC.TO ACUTE G.E.	3	3	3	2	3	MILD-MOD ED-14	12	3	0.749	102	2	144	1	8	11.2	72.00	6.10	1.20	1.00
31	5621315	1	42	1	24.00	ACUTE GASTROENTERITIS	5	3	2	2	3	MILD-MOD ED-15	4	2	3.170	100	2	60	2	2	10.3	64.00	4.56	1.10	0.80
32	966639	1	46	1	25.00	AKI	4	4	3	3	3	MILD ED-17	2	2	15.810	727	2	24	3		11.0	3.11	10.00	1.40	0.55
33	974262	1	46	1	24.20	URINARY TRACT INFECTION	4	4	3	3	3	MILD-MOD ED-16	2	2	15.580	624	2	72	1	3	12.5	2.12	5.87	0.90	0.60
34	5438533	1	43	1	20.20	ACUTE GASTROENTERITIS	4	3	3	4	3	MILD ED-17	6	1	4.820	268	2	6	3		10.2	1.08	2.80	0.60	0.80
35	975487	1	38	1	28.80	IHD-NSTEMI	4	4	4	3	4	MILD ED-19	8	1	15.700	111	2	48	1	2	11.5	2.87	10.70	0.85	0.84
36	979322	2	55	1	19.60	IHD-DCM-LVEF-35%	3	2	2	2	3	MILD-MOD ED-12	10	3	4.050	224	1	120	2	5	11.8	1.71	4.90	1.20	0.88
37	979394	2	51	1	24.60	ACUTE PANCREATITIS	4	3	2	2	3	MILD-MOD ED-14	4	1	11.290	628	2	96	1	5	10.3	1.84	13.40	0.58	1.40
38	986424	1	27	1	25.91	ABSCESS	5	3	3	4	5	NO ED-22	1	2	21.990	854	2	4	3		17.5	3.17	7.60	0.91	1.50
39	955626	2	57	1	19.10	IHD-NSTEMI	3	3	3	3	3	MILD-MOD ED-15	6	1	13.270	567	2		3		12.5	1.93	6.90	0.93	1.12
40	892140	1	42	1	22.40	VIRAL FEVER	4	4	3	4	3	MILD ED-18	8	1	7.850	282	2	72	1	2	9.4	2.06	5.80	0.80	0.44
41	967913	1	49	1	19.60	FEVER WITH THROMBOCYTOPENIA	3	2	2	2	2	MODERATE ED-11	12	3	0.154	30	2	96	1	5	9.6	3.20	6.20	1.00	0.80
42	1010383	1	49	1	24.30	CHRONIC DIARRHOEA	4	4	3	3	3	MILD-MOD ED-14	9	1	10.520	336	2	120	1	6	13.9	2.71	8.30	0.81	1.50
43	981840	1	42	1	23.60	IHD-NSTEMI	4	3	3	2	3	MILD-MOD ED-15	10	3	5.640	338	2	48	1	2	17.0	1.96	9.30	1.50	1.40
44	975629	1	35	1	23.40	ACUTE GASTROENTERITIS	5	5	4	4	4	NO ED-22	3	1	10.470	565	2		3		12.6	4.97	15.60	1.14	0.26
45	982320	1	47	1	27.70	IHD-NSTEMI	4	4	3	3	3	MILD ED-17	6	2	16.910	652	2	108	1	5	8.5	3.74	9.90	1.28	0.13
46	985921	1	48	1	25.50	LMN FACIAL PALSY	4	4	3	3	3	MILD ED-17	8	2	8.870	676	2	60	1	3	14.9	2.76	13.20	0.70	2.75
47	985368	1	47	1	24.20	AKI SEC.TO ACUTE G.E.	3	2	2	2	2	MODERATE ED-11	14	3	1.900	268	2	96	1	5	8.4	1.91	8.30	2.50	0.26
48	983537	1	43	1	24.60	FEVER WITH THROMBOCYTOPENIA	4	4	3	3	4	MILD ED-18	2	2	21.670	682	2	60	1	2	15.0	83.00	5.50	0.75	2.77
49	981485	2	52	1	22.40	VIRAL FEVER	3	3	3	3	3	MILD-MOD ED-16	6	1	2.180	445	2	120	1	5	8.8	54.00	4.80	0.97	0.57
50	984998	2	55	1	28.20	ACUTE GASTROENTERITIS	4	3	3	3	3	MILD-MOD ED-16	2	2	12.020	571	1	144	1	8	10.8	82.00	4.00	1.00	0.43

51	987143	1	47	1	27.60	ACUTE PANCREATITIS	5	4	4	4	4	MILD ED-21	1	2	14.760	850	2	120	1	8	14.2	3.30	10.30	0.88	1.57
52	925214	1	46	1	25.50	LEFT LOWER LIMB DVT	4	3	3	3	3	MILD-MOD ED-16	12	3	6.620	128	2	96	1	5	15.5	6.62	3.90	0.66	0.60
53	953040	2	56	1	25.00	BPH	4	3	2	3	3	MILD-MOD ED-15	14	3	9.830	776	2	120	1	8	13.7	6.93	6.93	0.84	1.30
54	983308	1	44	1	25.90	VIRAL FEVER	4	3	2	2	3	MILD-MOD ED-14	6	1	5.500	258	2	96	1	6	9.6	86.00	3.00	0.45	0.97
55	903635	1	30	1	25.10	VIRAL FEVER	3	3	2	2	3	MODERATE ED-13	12	3	2.600	192	2	12	3		9.8	3.04	7.79	0.80	0.38
56	1001144	1	23	1	26.20	VIRAL FEVER	4	3	4	4	4	MILD ED-19	2	2	28.300	752	2	12	1	1	14.5	2.74	7.20	0.92	1.20
57	993818	1	42	1	23.20	CELLULITIS	4	4	3	3	3	MILD ED-17	6	2	8.550	358	2	72	1	4	11.8	2.72	10.20	0.68	1.20
58	954870	2	52	1	19.20	COPD	3	3	2	3	3	MILD-MOD ED-14	14	3	1.360	158	1	120	2	7	7.5	3.70	5.19	0.70	0.60
59	1014651	1	40	1	24.10	HIV GASTROPATHY	4	4	3	4	4	MILD ED-19	2	1	11.780	567	2	96	1	5	15.0	1.97	5.20	0.84	0.55
60	988969	1	21	1	19.10	VIRAL FEVER	3	3	2	3	3	MILD-MOD ED-14	6	1	3.040	213	2	120	1	8	10.6	4.56	9.93	0.80	0.90
61	978814	1	40	1	19.20	ACUTE GASTROENTERITIS	3	3	3	3	3	MILD-MOD ED-15	8	1	3.170	180	2	96	1	2	8.0	2.93	8.19	1.50	1.20
62	1006492	1	45	1	18.60	LMN FACIAL PALSY	3	3	3	3	3	MILD-MOD ED-15	8	1	3.240	156	2	72	2	2	15.8	2.52	8.30	0.53	0.67
63	955386	2	56	1	20.50	DYSPESIA	4	3	4	4	3	MILD ED-18	4	2	9.090	660	2	144	1	8	11.0	1.91	4.00	0.91	0.92
64	937331	1	46	1	24.30	CELLULITIS	3	3	3	3	3	MILD-MOD ED-15	10	3	3.070	227	2	96	1	4	9.3	4.53	14.30	1.40	0.32
65	977974	1	47	1	23.80	CATARACT	3	2	2	3	2	MILD-MOD ED-12	14	3	0.210	128	2	120	1	5	14.0	3.82	9.20	1.20	0.90
66	1015307	1	49	1	21.20	ACUTE PANCREATITIS	4	3	3	4	4	MILD-MOD ED-16	8	1	12.850	468	2	144	2	7	11.0	1.98	8.40	0.63	1.20
67	927386	1	47	1	23.00	PANCYTOPENIA	3	3	2	4	4	MILD-MOD ED-15	10	3	3.520	166	1	120	1	8	7.8	1.60	7.40	1.19	0.60
68	966061	2	55	1	25.50	ACALCULOUS CHOLECYSTITIS	4	3	2	4	2	MILD-MOD ED-14	12	3	1.810	256	2	120	1	6	13.2	1.53	11.20	0.95	0.45
69	1007885	1	49	1	18.60	COPD	3	3	2	3	3	MILD-MOD ED-14	14	3	1.190	126	2	120	1	4	8.3	1.68	5.50	0.99	1.20
70	5460814	1	45	1	21.20	ACUTE GASTROENTERITIS	4	4	4	5	4	MILD ED-21	10	3	2.490	236	1	96	1	6	12.6	89.00	4.90	0.79	1.40
71	966251	1	48	1	24.60	AKI	3	3	2	4	2	MILD-MOD ED-14	8	3	10.460	213	2	96	1	6	10.6	2.48	9.80	1.40	0.40
72	964868	1	44	1	28.60	ACUTE GASTROENTERITIS	4	4	4	4	4	MILD ED-20	1	2	22.720	829	2	72	1	4	11.0	1.78	6.37	1.20	0.70
73	978958	2	58	1	26.30	IHD	4	4	3	4	2	MILD ED-17	4	2	13.270	348	1	192	1	12	12.5	1.93	6.90	0.93	1.20
74	927504	2	52	1	21.60	ACALCULOUS CHOLECYSTITIS	4	4	4	4	3	MILD ED-19	1	2	23.370	756	2	120	1	4	8.9	1.82	9.57	1.40	0.30
75	1010171	1	40	1	19.60	PANCYTOPENIA	3	3	3	3	3	MILD-MOD ED-12	6	1	5.510	270	2	96	1	4	7.5	1.08	1.28	0.50	1.20

SERIAL NO.	Direct bilirubin	SGOT	SGPT	Cholesterol	LDL	HDL	Triglycerides	USG abdomen	CXR
1	0.40	13	13	152	82	28	212	GRADE 1 RPC	1
2	0.15	20	76	192	63	96	167	PROSTATOMEGALY	1
3	0.10	11	13	186	84	42	186	GRADE 1 RENAL PARENCHYMAL CHANGES	1
4	0.05	18	68	92	52	26	102	MILD HEPATOMEGALY AND SPLENOMEGALY	1
5	0.22	14	35	136	62	34	112	N	1
6	0.20	60	40	156	92	44	96	ACALCOULOUS CHOLECYSTITIS	1
7	0.20	21	18	86	48	21	92	MILD SPLENOMEGALY	1
8	0.60	18	14	180	86	32	124	FATTY LIVER	1
9	0.80	32	16	102	42	22	94	FATTY LIVER,SPLENOMEGALY	1
10	0.30	82	42	142	66	38	120	N	1
11	0.40	12	16	148	68	40	110	FATTY LIVER,ENLARGED PROSTATE	1
12	0.10	29	10	82	46	12	96	N	1
13	0.08	25	20	122	58	28	178	N	1
14	0.50	10	11	129	80	24	123	ACUTE PANCREATITIS	1
15	0.10	24	34	163	99	44	99	N	1
16	1.00	126	19	83	49	14	101	GRADE 1 RPC	1
17	0.08	14	10	126	58	36	120	FATTY LIVER,RIGHT SMALL KIDNEY,GRADE 2 RPC	1

18	0.40	18	12	136	62	38	160	CHOLELITHIASIS	1
19	0.10	28	15	126	39	31	280	MILD HEPATOMEGALY AND SPLENOMEGALY	1
20	0.20	14	16	136	47	22	126	N	1
21	0.35	12	14	122	38	28	120	MILD SPLENOMEGALY	1
22	0.20	12	5	116	62	28	96	MILD SPLENOMEGALY,MILD ASCITES	1
23	0.20	19	16	122	64	36	122	GRADE 1 RPC	1
24	0.25	35	18	101	60	23	88	HEPATOSPLENOMEGALY,CYSTITIS,	1
25	0.14	70	16	72	24	36	59	MILD SPLENOMEGALY,MILD ASCITES	1
26	0.40	13	23	96	26	38	86	N	1
27	0.40	18	12	142	72	36	124	MILD SPLENOMEGALY	1
28	0.12	10	33	170	105	23	52	N	1
29	0.10	51	60	128	64	32	120	N	1
30	0.60	32	16	92	45	16	155	HEPATOMEGALY WITH FATTY LIVER,GRADE 1 RPC	1
31	0.20	56	18	118	36	24	106	HEPATOSPLENOMEGALY,CYSTITIS,	1
32	0.22	11	13	110	42	22	96	GRADE 1 RENAL PARENCHYMAL CHANGES	1
33	0.20	18	22	148	44	32	126	N	1
34	0.30	37	20	124	34	22	100	FATTY LIVER	1
35	0.38	18	16	118	66	34	89	MILD HEPATOMEGALY AND FATTY INFILTRATION	1
36	0.39	19	53	92	22	32	72	N	1

37	0.67	45	62	172	113	25	168	FATTY LIVER,ACUTE PANCREATITIS	1
38	0.44	33	28	166	96	38	144	FATTY LIVER	1
39	0.26	23	25	136	48	32	120	MILD SPLENOMEGALY	1
40	0.15	12	10	128	34	28	110	MILD SPLENOMEGALY,MILD ASCITES	1
41	0.30	22	26	92	22	24	126	MILD HEPATOMEGALY AND SPLENOMEGALY	1
42	0.39	29	25	157	101	35	106	N	1
43	0.60	77	138	149	82	32	110	GRADE 1 RPC	1
44	0.20	23	25	148	76	32	112	MILD HEPATOMEGALY AND FATTY INFILTRATION	1
45	0.07	60	32	158	58	28	96	GRADE 1 RPC	1
46	1.20	34	20	135	68	43	122	MILD HEPATOMEGALY AND SPLENOMEGALY	1
47	0.08	28	21	80	11	20	247	MILD HEPATOMEGALY AND FATTY INFILTRATION,GRADE 1 RPC	1
48	0.48	45	36	160	74	42	148	N	1
49	0.23	89	72	109	59	21	147	HEPATOMEGALY WITH FATTY LIVER	1
50	0.03	13	37	69	14	40	46	N	1
51	0.21	26	24	148	52	42	110	MILD HEPATOMEGALY AND ACUTE PANCRATITIS	1
52	0.20	31	57	135	60	40	176	N	1
53	0.40	12	18	199	116	38	227	FATTY LIVER,ENLARGED PROSTATE	1
54	0.70	84	73	136	58	36	102	FATTY LIVER,SPLENOMEGALY	1
55	0.13	45	42	156	64	32	120	N	1

56	0.50	22	18	196	72	34	132	MILD HEPATOMEGALY AND SPLENOMEGALY	1
57	0.60	16	28	158	42	28	110	N	1
58	0.20	21	7	110	32	26	92	MILD HEPATOMEGALY AND SPLENOMEGALY	1
59	0.22	58	57	262	122	117	116	N	1
60	0.40	53	12	122	32	24	110	MILD HEPATOMEGALY AND FATTY INFILTRATION	1
61	0.70	26	9	126	42	32	96	HEPATOSPLENOMEGALY,CYSTITIS,	1
62	0.03	16	39	155	73	63	93	N	1
63	0.37	12	38	178	72	64	124	FATTY LIVER	1
64	0.16	13	12	126	44	34	98	N	1
65	0.50	18	14	105	44	21	201	CHOLELITHIASIS	1
66	0.80	32	22	124	42	32	78	N	1
67	0.10	30	33	146	32	26	110	HEPATOSPLENOMEGALY,CYSTITIS,	1
68	0.20	28	19	83	53	12	88	N	1
69	0.50	40	29	120	46	24	110	FATTY LIVER,SPLENOMEGALY	1
70	0.50	80	33	137	65	48	122	N	1
71	0.20	30	22	156	62	42	96	GRADE 1 RPC	1
72	0.40	26	12	138	46	58	170	N	1
73	0.60	22	10	128	59	55	59	CHOLELITHIASIS	1
74	0.10	32	40	196	72	54	130	ACALCOULOUS CHOLECYSTITIS	1

75	0.50	40	30	110	22	32	82	N	1