PREVALENCE OF ERECTILE DYSFUNCTION AND ASSOCIATED RISK FACTORS AMONG DIABETIC MEN ATTENDING DIABETIC CLINIC AT MUHIMBILI NATIONAL HOSPITAL IN DAR-ES-SALAAM, TANZANIA

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PREVALENCE OF ERECTILE DYSFUNCTION AND ASSOCIATED RISK FACTORS AMONG DIABETIC MEN ATTENDING DIABETIC CLINIC AT MUHIMBILI NATIONAL HOSPITAL IN DAR ES SALAAM, TANZANIA

By

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A dissertation Submitted in (partial) Fulfillment of the Requirements for the Degree of Master of Medicine (Internal Medicine) of Muhimbili University of Health and Allied Sciences

Muhimbili University of Health and Allied Sciences

September, 2012

CERTIFICATION

The undersigned certify that they have read and hereby recommend for examination of dissertation entitled ***Prevalence of Erectile dysfunction and associated risk factors among diabetic men attending diabetic clinic at Muhimbili National Hospital in Dar Es Salaam, Tanzania,*** in fulfillment of requirements for the degree of Master of Medicine (Internal Medicine) of Muhimbili University of Health and Allied Sciences.

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CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by Muhimbili University of Health and Allied Sciences of dissertation entitled ***Prevalence of Erectile dysfunction and associated risk factors among diabetic men attending diabetic clinic at Muhimbili National Hospital in Dar Es Salaam, Tanzania,*** in fulfillment of requirements for the degree of Master of Medicine (Internal Medicine) of Muhimbili University of Health and Allied Sciences.

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Prof. J. Lutale

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I, **Reuben Mutagaywa**, declare that this **dissertation** is my own original work and that it has not been presented and will not be presented to any other university for a similar or any other degree award.

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DEDICATION

This dissertation is dedicated to my dear wife, Aileen and my mother Zeulia. It is also dedicated to the memory of my late father, Paulo Samwel Mutagaywa, who inspired me right from childhood to study hard and become a good doctor.

ABSTRACT

Introduction: There has been an increase in the prevalence of erectile dysfunction (ED) in the general population. This increase has been attributed to by several factors. Among these factors Diabetes Mellitus have been shown to be a major determinant.

Objective: To determine the prevalence of erectile dysfunction and associated risk factors among diabetic men attending diabetic clinic at Muhimbili National Hospital (MNH).

Methodology: This was a prospective cross-sectional hospital based study, was conducted among 312 consecutively recruited diabetic male patients (Mean age = 51.33 years, SD = 15.03 years) for the period of 8 months from May to December 2011. Data were collected using a structured questionnaire, which consisted of several demographic variables, clinical aspects variables and International Index of Erectile Function (IIEF) questionnaire.

Descriptive statistics were calculated to describe the study population and check for initial associations at univariate level. Multivariate logistic regression was used to identify and quantify predictors of ED among these men. Variables that were entered in the equation were duration of diabetes mellitus (DM), type of DM, history of smoking, history of alcohol, history of stroke, history of peripheral neuropathy, monofilament score, being hypertensive, lipid profile, fasting blood sugar, level of Glycosylated hemoglobin (HbA1c) and Ankle Brachial Index (ABI) score while controlling for other demographics.

Results: The prevalence of erectile dysfunction was found to be 55.1% (n= 172). Forty patients (12.8%) had mild dysfunction, 45 (11.5%) had moderate dysfunction, and 87 (27.9%) had severe dysfunction.

The prevalence was higher (78%) in men aged 60 years and above ($p < 0.001$); there is a trend towards increase in prevalence of erectile dysfunction as the age increases. Longer duration of DM, type 2 DM, history of peripheral neuropathy, history of hypertension, low score on monofilament test, and evidence of peripheral vascular disease were highly significantly associated with ED ($p =<0.001$). History of smoking, history of drinking alcohol, and history of stroke were also significantly associated ED ($P = 0.02$, 0.05, and 0.01 respectively). On multivariate logistic regression the predictors of ED were found to be old age (odds ratio [OR]

= 7.1, 95% CI 1.2-40.7), evidence of peripheral neuropathy ([OR] =5.9, 95% CI 1.6-21.3), and evidence of peripheral vascular disease ([OR] =2.5, 95% CI 1.2-5.3) {p= 0.028, 0.019, and 0.007 respectively}. This study also showed that ED was highly statistically significant associated with other sexual domains (p <0.001).

Conclusions and Recommendations: This study showed that the prevalence of erectile dysfunction is high among DM patients. Interventions aimed at prevention of DM especially type 2; early diagnosis of DM and detection of its complications, and adherence to treatment to prevent complication should be implemented. Further studies should emphasize on temporal variation to show true causality of DM on erectile dysfunction especially in low-income settings.

ABBREVIATIONS

BMI	-	Body Mass Index
BP	-	Blood Pressure
CVD	-	Cardiovascular Disease
DIED	-	Diabetic -Induced Erectile Dysfunction
DM	-	Diabetes Mellitus
ED	-	Erectile Dysfunction
FPG	-	Fasting Plasma Glucose
HDL	-	High Density Lipoprotein
IDDM	-	Insulin Dependent Diabetic Mellitus
IIEF	-	International Index of Erectile Function
MMAS	-	Massachusetts Male Aging Study
MNH	-	Muhimbili National Hospital
MOI	-	Muhimbili Orthopaedic Institute
MUHAS	-	Muhimbili University of Health and Allied Sciences
NIDDM	-	Non Insulin Dependent Diabetic Mellitus
TATAKI	-	Taasisi ya Taaluma ya Kiswahili
T1DM	-	Type 1 Diabetes Mellitus
T2DM	-	Type 2 Diabetes Mellitus
TG	-	Triglyceride
USA	-	United States of America
VLDL	-	Very Low Density Lipoprotein
WHO	-	World Health Organisation

DEFINITION OF TERMS

Sexual dysfunction refers to a problem during any phase of the sexual response cycle that prevents the individual or couple from experiencing satisfaction from the sexual activity. The sexual response cycle has four phases: excitement, plateau, orgasm, and resolution.

Erectile dysfunction (ED), which is also referred to as impotency in males, is defined as the inability to achieve and/or maintain an erection sufficient to permit satisfactory sexual intercourse.

Sexual activity includes intercourse, caressing, foreplay and masturbation

For the purpose of this study, sexual intercourse was defined as vaginal penetration of the partner

Sexual stimulation includes situations like foreplay with a partner, looking at erotic pictures, etc.

Ejaculate is defined as the ejection of semen from the penis (or the feeling of this).

Sexual desire (libido) is defined as a feeling that may include wanting to have a sexual experience (for example, masturbation or intercourse), thinking about having sex or feeling frustrated due to a lack of sex.

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CHAPTER ONE

1.0 INTRODUCTION

1.1 Background

Sexual dysfunction refers to a problem during any phase of the sexual response cycle that prevents the individual or couple from experiencing satisfaction from the sexual activity. The sexual response cycle has four phases: excitement, plateau, orgasm, and resolution.

Erectile dysfunction (ED) is defined as inability to achieve and maintain an erection sufficient to permit satisfactory sexual intercourse[1].

The causes of ED are numerous but generally fall into two categories, organic or psychogenic. The organic causes can be subdivided into five categories: vascular, traumatic/post surgical, neurological, endocrine-induced, and drug-induced. Examples of the psychogenic causes are depression, performance anxiety, and relationship problems[2].

A variety of chronic illnesses such as diabetes mellitus, cardiovascular disease and depression are associated with higher rates of ED[3]. Diabetes mellitus appears to be a major determinant of ED[4]. Diabetic men are 3 times as likely to develop ED as non-diabetic men[5]. Increased duration of diabetes increases both the rate and severity of ED[6]. It can occur early in the course of the disease and it can occasionally be the presenting symptom[7].

Main risk factors of ED in people with diabetes are neuropathy, vascular insufficiency, poor glycemic control, hypertension, low testosterone levels, and lifestyle factors such as smoking, alcohol and inactivity. In addition the prevalence of ED greatly increases with age [8-11].

A wide range of prevalence rates of ED among diabetic men has been reported in various studies. These studies suggest that its prevalence in men with diabetes ranges from 35–75% versus 26% in general population. Variation in prevalence is mainly due to differences on definitions used, differences in the populations studied, study design, classification, and the

diagnostic tools- especially for studies conducted in the past. Introduction of validated questionnaire (the IIEF) minimized this variation[2].

Many male sexual function profiles and ED questionnaires have been developed. The International Index of Erectile Function Questionnaire (IIEF) is the most reliable measure of ED [3, 12]. It has 15 questions and the total score is obtained by the sum of the individual scores of each question. It addresses and quantifies five domains, namely: erection function, orgasmic (ejaculation) function, sexual desire (libido), intercourse satisfaction (ability to sustain intercourse), and overall satisfaction/premature ejaculation.

Although other diagnostic procedures such as medical and sexual history are useful,[13, 14] it has been proposed that ED, is best assessed by means of patients self report technique[13]

The IIEF questionnaire has demonstrated high specificity and sensitivity for detecting changes in erectile function. It discriminates well between men with and without ED and has been used to assess levels of ED severity, therapeutic efficacy and outcomes in patients with ED [15, 16]

In this study therefore, the author looks and uses the IIEF in analyzing domains of sexual function in respect to severity in patients with DM. The main objective was to determine the prevalence of ED in diabetic male patients and also the associated factors.

Two other questionnaires have also been used in clinical studies- The Sexual Encounter Profile (SEP) and the Global Assessment Questionnaire (GAQ) [12]. These questionnaire are superseded by the IIEF.

Though there is no single treatment plan for ED, initial preventive measures helps to reduce the risk of developing ED. Effective new treatments are now available, including oral medications, injectable drugs, vacuum devices, and inflatable prostheses.

1.2 Historical aspects

The first description of ED dates back from about 2000 BC and was set down on Egyptian, Papyrus. Two types were described, natural impotence (the man is incaple of accomplishing the sex act) and supernatural impotence (from evil charm and spells) [17]. Later, Hippocrates described many cases of male impotence among rich inhabitants of Scythia and concluded that too much horse riding was the cause, the poor were not affected because they travel by foot[17]

The current understanding of the erectile physiology was elaborated between 1980s and 1990s. In addition to the role of smooth muscle in regulating arterial and venous flow, the three-dimensional structure of the tunica albuginea and its role in venous occlusion have been elucidated. An important breakthrough in the understanding of the neural control is the identification of nitric oxide (NO) as the major neurotransmitter for erection and of phosphodiesterase (PDEs) in returning the penis into a flaccid state[17] . The role of endothelium in regulating smooth muscle tone and the intercellular link by means of gap junctions has also been uncovered. Pathophysiologicaly, the changes of the smooth muscle, endothelium, and fibroelastic framework in patients with diabetes, atherosclerosis, and aging have also been identified [13, 17-19]

1.3 Functional anatomy of the penis

The penis is a pendulous organ suspended from the front and sides of the pubic arch and containing the greater part of the urethra. In the flaccid condition it is cylindrical in shape, but when erect it assumes the form of a triangular prism with rounded angles, one side of the prism forming the dorsum. It is composed of three cylindrical masses of cavernous tissue bound together by fibrous tissue and covered with skin. Two of the masses are lateral, and are known as the corpora cavernosa penis, the third is the median, and is termed the corpus cavernosum urethra[20]

The corpora cavernosa penis forms the greater part of the substance of the penis. For their anterior three-fourths they lie in intimate apposition with one another, but behind they diverge in the form of two tapering processes known as the crura, which are firmly connected to the

rami of the pubic arch. Traced from behind forward, each crus begins by a blunt-pointed process in front of the tuberosity of the ischium. Just before it meets its fellow it presents a slight enlargement, named by Kobelt the bulb of the corpus cavernosum penis. Beyond this point the crus undergoes a constriction and merges into the corpus cavernosum proper, which retains a uniform diameter to its anterior end. Each corpus cavernosum penis ends abruptly in a rounded extremity some distance from the point of the penis[20].

The corpus cavernosum urethra (corpus spongiosum) contains the urethra. Behind, it is expanded to form the urethral bulb, and lies in apposition with the inferior fascia of the urogenital diaphragm, from which it receives a fibrous investment. The urethra enters the bulb nearer to the upper than to the lower surface. On the latter there is a median sulcus, from which a thin fibrous septum projects into the substance of the bulb and divides it imperfectly into two lateral lobes or hemispheres[20].

The portion of the corpus cavernosum urethra in front of the bulb lies in a groove on the under surface of the conjoined corpora cavernosa penis. It is cylindrical in form of an obtuse cone, flattened from above downward. This expansion, termed the glans penis, is moulded on the rounded ends of the corpora cavernosa penis, extending further on their upper than on their lower surfaces. At the summit of the glans is a slit-like vertical external urethral orifice[20]

The circumference of the base of the glans forms a rounded projecting border, the corona glandis, overhanging a deep retroglandular sulcus behind which is the neck of the penis[20].

For descriptive purposes it is convenient to divide the penis into three regions : the root, the body, and the extremity[20].

1.3.1 Haemodynamics and mechanisms of erection and detumescence

1.3.1.1 Corpora carvenosa

The penile erectile tissue, specifically the carvenous smooth muscles of the arteriolar and arterial wall plays a key role in erectile process.

In flaccid state these smooth muscles are tonically contracted, allowing only a small amount of arterial flow for nutritional purposes. Sexual stimulation triggers release of neurotransmitters from the carvenous nerve terminals. This results in relaxation of these smooth muscles.

Erection thus involves sinusoidal relaxation, arterial dilatation and venous compression.

1.3.1.2 Corpus spongiosum and glans peni

During erection the arterial flow increases in similar manner as in corpora carvenosa, however the pressure in the corpus spongiosum and the glans is only one third to one half of that of corpora carvenosa because the tunica covering (thin over the corpus spongiosum and virtually absent over the glans) ensures minimal venous occlusion.

1.3.1.3 Smooth muscle physiology

Spontaneous contractile activity of carvenous smooth muscle has been recorded in vitro and in vivo. Mandrek (1994)[17] demonstrated spontaneous mechanical activity with a frequency of 6 to 30 contractions per minute accompanied by fluctuations in membrane potential stimulation of the tissue with tetraethylammonium chloride and norepinephrine produced strong tonic contractions with relative electrical silence

In the study of myosin isoforms in smooth muscle cells in the corpus carvenosum penis, Disanto and associates (1998) reported that the corpora carvenosum smooth muscle cells process an overall myosin isoform composition intermediate between aorta and bladder smooth muscles which generally expresses tonic and phasic like characteristics respectively

1.3.2 Blood supply

The main source of arterial supply to the penis is through the internal pudendal artery a branch of internal iliac artery

The venous drainage from the three corpora originates in tiny venules leading from the peripheral sinusoids immediately beneath the tunica albuginea.

1.3.3 Nerve supply

Innervations of penis is both autonomic (sympathetic and parasympathetic) and somatic (sensory and motor)

The sympathetic pathway originates from the 11^{th} thoracic to 2^{nd} lumbar spinal segments and passes through the white rami to the sympathetic chain ganglia. Some fibers then travel through the lumbar splanchnic nerves to the inferior mesenteric and superior hypogastric plexus, from which fibers travel in the hypogastric nerves to the pelvic plexus.

In humans the T10 and T12 segments are most often the origin of the sympathetic fibers and the chain ganglia cells projecting to the penis are located in sacral and caudal ganglia[21].

Parasympatic pathways arise from the neurons in the intermediolateral cell columns of the second third and fourth sacral spinal cord segment.

The carvenous nerves are branches of the pelvic plexus that innervates the penis. Stimulation of the pelvic plexus and carvenous nerve induces erection whereas stimulation of the sympathetic trunk induces detumescence.

The somatosensory pathway originates at the sensory receptors in the penile skin, glans and urethra and within the corpus carvenosum.

The nerve fibers from the receptors converge to form bundles of the dorsal nerve of the penis, which joins otther nerves to become the pudendal nerve. The latter enter the spinal cord via the S2-S4 roots to terminate on the spinal neurons and interneurons in the central gray region of the lumbosacral region. Activation in these sensory neurons sends messages of pain, temperature and touch by means of spinothalamic and spinorectal pathways to the thallamus and sensory cortex for sensory perception.

Contraction of the ischiocarvenous muscles produces the rigid erection phase. Rhythmic contraction of the bulbocarvenosus muscle is necessary for ejaculation.

1.4 Pathophysiology of penile erection

Numerous factors can disrupt the normal physiologic mechanisms involved in erection. Most cases of erectile dysfunction were thought to be psychologically based, but it is now understood that most have an organic cause, especially in older patients. In about 40% of men over 50years old, the primary cause of erectile dysfunction is related to atherosclerotic disease[22]. In this respect, risk factors are similar to those of cardiovascular disease. However, ED may also be related to psychological, neurological, hormonal, pharmacological and end organ (penile) factors. With respect to neurological causes, involvement of any nerve group, either central or peripheral, may lead to ED. Cerebral diseases lead to decreased sexual interest, possibly through over-inhibition of spinal centers. Among patients with spinal cord injury, 95% of those who have upper motor neuron lesions are capable of reflexogenic erections, 25% of patients who have lower motor neuron lesions are capable of psychogenic erections, and more than 90% of patients who have incomplete lesions either kind retain their erectile function. Direct injury to the carvenosal nerve and blood supply commonly occurs during therapy for prostate cancer; ED is present to some extent in 80% of patients so treated, whether by surgery or external beam radiation therapy.

Almost all antihypertensive medications have been implicated in ED[23]. The mechanisms vary from central-acting sympatholytics, depression of libido as well as higher blood pressure requirements to achieve erection in atherosclerotic patients taking diuretics and venodilators. The role of smoking in causing ED remains a source of controversy. A recent consensus group analysis failed to document a direct link to ED. However, smoking seems to amplify other risk factors such as hypertension and coronary artery disease[24].

Martin-Morales and colleagues[25] have shown an increased risk of ED among smokers (OR=2.5). Systemic diseases are also associated with ED. This is often multifactorial, as in diabetes and renal failure. Given the role of androgens in physiology of erections, it is not surprising that many hypogonadal men do not experience ED per se. Low serum of testosterone levels may be primary or secondary to another condition, such as hyperprolactinemia or hypothyroidism. Regardless of the mechanism, the result is primarily a loss of libido, although many hypogonadal men retain their ability to obtain erections.

CHAPTER TWO

2.0 LITERATURE REVIEW, PROBLEM STATEMENT, RATIONALE, AND OBJECTIVE

2.1 LITERATURE REVIEW

2.1.1 Prevalence of ED and its associated factors in the general population

Erectile dysfunction increases with age. According to the Massachusetts Male Aging Study (MMAS), which surveyed 1790 men, the prevalence of ED was approximately 52% in men aged 40 to 70 [5] . It was estimated that in 1995 there were over 152 million men worldwide who experienced erectile dysfunction. With ageing worldwide population, it has been projected that by the year 2025, approximately 322 million men will have some degree of erectile dysfunction. The largest projected increases were in the developing world i.e. Africa, Asia and South America. The projected trend represents a serious challenge for health care policy makers to develop and implement policies to prevent or alleviate ED [26, 27].

In another study done in U.S.A [5] among 31742 men aged 53 to 90 years, the age-specific prevalence of ED was 33%. Many aspects of sexual function decreased sharply by decade after 50 years of age. Several modifiable behaviors were associated with maintenance of good erection function, even after co-morbid conditions were considered. Lifestyle factors mostly associated with ED were physical activity and leanness.

Bai et al [28] determined the age-adjusted prevalence of ED in 3 big cities of China and explored its potential sociodemographic, medical and lifestyle correlates. The age-adjusted prevalence of ED was 28.34% (mild 15.99%, moderate 7.14%, and severe 5.21%). The prevalence of ED increased with age. Cardiovascular disease, diabetes and hyperlipidaemia were positively correlated with the increased prevalence. Sociodemographic and lifestyle factors, such as education, spouse companionship, living conditions, cigarette and alcohol consumption or duration also have association with prevalence of ED.

In a large cross-sectional study done in Singapore by Tan et al [29], the overall prevalence of erectile dysfunction was 51.3% and prevalence rate increased significantly with age. Age group sub-analysis (40 to 69 years) gave a prevalence rate of 52.7% which was very similar to that reported in the Massachusetts Male Ageing Study (52%) despite a very different sample population-mainly Caucasians and utilizing a different research instrument and methodology. While it is not possible to do direct comparison due to different methodology and sampling technique, other studies gave lower prevalent rate of erectile dysfunction than that reported in Singapore study with Thailand [30] (37.5% of men 40-70 years), Australia [31] (33.9% of men 40-69 years) and Italy [32] (overall prevalence of ED was 12.8%)

In a population-based study done in Casablanca, Morocco by Berrada et al [33], ED was prevalent in 54%, increased with age, and was highly prevalent in both the illiterate and those employed. Risk factors were diabetes, hypertension, heart disease, and smoking.

In Nigeria, the prevalence of ED among patients attending primary care clinics, age-standardized to U.S. population in 2000 was 57.4%. This high ED prevalence was associated with etiologies such as psychosexual factors, chronic medical conditions, and some lifestyle [34].

In order to expand cross-cultural understanding of erectile dysfunction Gray, P et al [35] investigated ED among Ariaal men, pastoral nomads of northern Kenya. It was revealed that ED increases with age with men 60years and older showing significantly higher ED compared with men in their 20years, 40years and 50years. In addition, there was a significant interaction between age group and marital status.

In Tanzania, a study done by YN Kapona and CS Yongolo on assessing male erectile dysfunction as seen in Dar-es-Salaam City over a period of three years January 2001 to December 2003, 4060 patients were seen at reproductive health clinic out of these two hundred and three patients (5%) had ED [36].

As pointed out previously in different epidemiological studies, ED increases with advancing age sharing the same risk factors related to atherosclerosis such as hypertension,

dyslipidaemia, smoking, and diabetes [5, 37]. All these co-morbidities are also related to endothelial dysfunction which plays a central role in the pathophysiology of ED as well as in the development of atherosclerosis and its complications [38, 39].

2.1.1.1 Prevalence of ED in patients with hypertension and other cardiovascular disease (CVD)

Recent analyses suggest that about 67-68% of men with hypertension have some degree of erectile dysfunction. With about 25million men in the U.S with hypertension, substantial numbers of hypertension-related ED exist that tend to be of a more severe nature than the general population. Men with hypertension are more likely to have ED. Thiazide diuretic and beta-blocker therapy are more likely to contribute to ED [40], but almost all antihypertensive medications have been implicated in erectile dysfunction [23]. The mechanisms vary from central-acting sympatholytics, depression of libido as well as higher blood pressure requirement to achieve erection in atherosclerotic patients taking diuretics and vasodilators.

Erectile dysfunction is also a common problem in male patients with heart failure (HF). The overall prevalence of ED by U.S ethnic groups (Hispanic, Black and White) with HF was 89% and ED severity did not vary by race/ethnic group [41].

2.1.1.2 Prevalence of ED in patients with chronic renal failure

Cerqueira, J et al [42] conducted a study to determine the prevalence of ED and associated variables in patients with chronic renal failure(CRF). The prevalence of ED in this population was 57.9%.

Another study [43] done among patients with End Stage Renal Disease (ESRD) recipients of kidney transplant showed ED in 48.9% (89 recipients). Patients with ESRD experience disturbances in erectile function related to organic factors such as uremia, hypertension, endocrine, and nonorganic factors like depression.

2.1.1.3 Dyslipidaemia and ED

Wei, M et al [44] reported the relationship between serum cholesterol and erectile dysfunction. Men with HDL-chol measurement over 1.55mmol/l (60mg/dl) had 0.30 times the risk (95% CI 0.09-3.03) as did men with less than 0.78mmol/l (30mg/dl). Men with total cholesterol over 6.21mmol/l (240mg/dl) had 1.83 times the risk (95% CI 1.00-3.37) as did men with less than 4.65mmol/l (180mg/dl). Those differences remained essentially unchanged after adjustment for other potential components.

2.1.1.4 Cigarette Smoking and ED

Smoking is associated with an increased risk of developing ED. In the MMAS, cigarette smoking amplified the risk of ED associated with other risk factors (e.g. Hypertension, diabetes and dyslipidaemia) or with ageing. At the molecular level, several changes associated with smoking have been documented in man and experimental model. These changes could contribute to the pathogenesis of ED. Furthermore, studies performed in dogs demonstrated a smoking- related reduction in arterial blood flow and venous restriction. Similarly, impaired penile flow has been documented in men who smoke [24, 45].

Martin- Morales and colleague[s] [25] have shown an increased risk of erectile dysfunction among smokers (odds ratio OR= 2.5).

2.1.1.5 Lower Urinary Tracts Symptoms (LUTs) and ED

In a prospective study done in Asia by Wong et al [46] on the association between LUTs and ED, a dose-response relationship was observed for the relationship between severity of LUTs and severity of ED .Those who had more severe LUTs had an increased odd of having more severe ED (OR (95CI): 1.88(1.16-2.97) for mild LUTs; 2.95 (1.81-4.81) for moderate LUTs ; and 3.82,(2.00-7.27) for severe LUTs. Other factors that were statistically significant associated with ED included BMI, HTN and DM.

In his M.Med dissertation case control study of sexual dysfunction in patients with lower urinary tract symptoms at Muhimbili National Hospital, Mbalamula Athuman [47] found the magnitude of ED to be significantly higher in patients with LUTS (84.4%) compared to control group- those without LUTS (56%), p value=0.008.

2.1.1.6 Metabolic syndrome (MS) and ED

Data available in literature support an association between MS and ED, but a clear cause-effect relationship has not yet been provided. The association between MS and ED was first described by Gunduz etal [48] in 2004 and Esposito et al [49] in 2005. Its role as an independent risk factor for ED was reinforced by Heidler et al [50] .

Corona et al [51] studied the prevalence of sexual dysfunction associated with the MS over a group of 803 male patients. Among the 230 patients diagnosed having MS, 96.5% reported ED.

2.1.1.7 Alcohol abuse and ED

Excessive alcohol intake has been shown to have an additive deleterious effect on diabetes. Some ED studies have found an association between a moderate consumption and better erection in both the general population [10, 52] and in men with diabetes [53, 54] .

However, another study showed a slightly higher prevalence of ED among alcohol consumers [55] . It is possible that moderate alcohol consumption exerts a protective effect on ED as appear to be the case for ischemic heart disease. However, most of these association studies are cross-sectional and should be interpreted with caution.

2.1.1.8 Human Immunodeficiency Virus and ED

As many as 7 out of 10 men with HIV have ED [56]. HIV infection has been linked to sexual dysfunction. This can be explained by the interactions between the reproductive system with the immune system, the endocrine, the neuroendocrine system and vascular system [57]. HIV

infection is also associated with a significant psychological impact, and furthermore long-term antiretroviral therapy has a negative psychological effect on sexual experience.

In his M.Med dissertation to determine th prevalence of ED and the associated risk factors among HIV patients in Dar es Salaam Care and Treatment Clinics in 2009, Eliasa Mkongo found a prevalence of 80% of ED among HIV patients[58].

2.1.1.9 Hypogonadism and ED

The prevalence of hypogonadism among men with ED varies from 1.7% [59] to 35% [60]. The extent of hypogonadism in men with ED, compared with men without ED remains unclear because most studies do not have adequate controls.

The strongest evidence of the role of testosterone in erection in humans come from studies of castrated men, 58% to 100% men develop complete or partial ED following castration [61]

2.1.2 Pathophysiology of Diabetic Induced Erectile Dysfunction (DIED)

The association between DM and ED was first documented in 1798 [62, 63]. Since that time, the etiology of ED in DM became clearer. The etiologies of ED in diabetic patients are multifactorial but essentially there are 3 mechanisms by which DM can produce ED: peripheral neuropathy, vascular disease, and hypogonadism.

It is well established that DM adversely affects not only peripheral nerve function, but also peripheral blood flow l [64] both of which have the potential to impede normal erectile function which is contingent upon tissue innervations and perfusion.

Atherosclerosis in the penile and pudendal arteries limits the blood flow into the corpus cavernosum. Because of the loss of compliance in the cavernous trabeculae, the venous flow is also lost. This loss of flow results in the inability of the corpora cavernosae to expand and compress the outflow vessel. Autonomic neuropathy is a major contributor to the high incidence of ED in people with diabetes. Norepinephrine- and acetylcholine-positive fibers in the corpus cavernosum have also been shown to be reduced in people with diabetes. This

results in loss of the autonomic nerve–mediated muscle relaxation that is essential for erections [2].

Hypogonadism is known to be a pathophysiologic factor in approximately 12% of the men who have ED both in diabetic and nondiabetic [65]. Normal erection requires a threshold level of testosterone level, and testosterone replacement is clearly indicated for men with hypogonadism and ED [66]. The relationship between DM and ED due to hypogonadism was explored by Stellato et al [67], who prospectively examined the association between low testosterone levels and sex hormone-binding globulin (SHBG) and the subsequent development of T2D in the cohort of the MMAS [67]. He also found that the end-organ damage secondary to hyperglycemia, as well as the co-morbidities in the patients and side-effects of the various medications (i.e. antihypertensives) they consume, all contribute to their ED.

At molecular level the proposed mechanisms of ED in diabetics include: elevated advanced glycation end-products (AGEs) and increased levels of oxygen free radicals, impaired nitric oxide (NO) synthesis, decreased and impaired cyclic guanosine monophosphate (cGMP)-dependent kinase-1 (PKG-1), increased endothelin B (ETB) receptor binding sites and ultrastructural changes, upregulated RhoA/Rhokinase pathway, and NO-dependent selective nitrergic nerve degeneration [18, 19]

2.1.3 Prevalence of ED and its associated factors in diabetes patients

Generally 25–75% of men with Type 2 diabetes complain of erectile dysfunction [68]. In numerous epidemiological studies the odd ratio of having erectile dysfunction if a man is diabetic is 1.9–4 times greater than a population without diabetes, making diabetes one of the greatest risk factors for erectile dysfunction [69].

Khatib, F.A et [70] al reported the overall prevalence of ED among Jordanian men with diabetes to be 62%, they found that 30.3% had severe ED. The prevalence increased with age from 26.5% in men aged under 40y, to 90% in age group above 70y. Severity of ED increased with age as well. Apart from age other independent risk factors of ED were hypertension, glycemic control, coronary artery disease, retinopathy, and neuropathy.

In 2003 in Italy, Mota etal [71] , studied 310 male diabetic patients and observed that ED showed a positive correlation with obesity, stroke, arteriopathy, retinopathy and smoking, but it was not correlated to type of DM, duration of diabetes <10yrs, hypertension, Ischemic heart disease, nephropathy, dyslipidaemia and alcohol consumption.

McCulloch et al [63] evaluated the frequency of ED in diabetes in a survey of 541 men aged 20 to 59 years with diabetes attending a large community clinic in West Germany. The prevalence of ED increased progressively with age, from 6% in men aged 20 to 24 years, to 52% in men aged 55 to 59 years. In addition to increasing age, the main risk factors associated with ED were peripheral or autonomic neuropathy, retinopathy, long duration of diabetes, and poor glycemic control.

In 1998 Fedele, D et al [72] analyzed the prevalence and risk factors for ED in men with IDDM and NIIDM in Italy. The prevalence of ED was found to be 35.8% and it increased with age, from 4.6% in men aged 20-29 to 45.5% in those aged ≥ 60years. Men with NIIDM reported less frequent ED than did men with IDDM. Men with good metabolic control had less ED as compared to those with poor control. Men with long duration of DM had higher risk of ED than those with short duration.

In the study done in by Balde, NM et al [73] in Conakry (Guinea), Erectile dysfunction concerned 90 patients (48%) of whom severe form was observed in 54%, moderate form in 35% and mild form in 12%.The patients who presented with ED were significantly older, displayed longer duration of diabetes with more complications (sensorial neuropathy and macroangiaopathy) and often took drugs for associated cardiovascular diseases. In 28% of the cases, erectile dysfunction was associated with a decline in libido and in 26% with ejaculation disorders.

In a cross-sectional study done by Ngalyuka et al in 2008 at Kenyatta National Hospital, ED was found to be five times more prevalent in diabetic males compared to age-matched controls with a prevalence of 74.3% and 15.1 % in diabetics and controls respectively. Advancing age, duration of diabetes, poor glycemic control, history of hypertension, use of antihypertensive

drugs, presence of peripheral neuropathy and evidence of peripheral arterial disease were associated with ED in male diabetics [74].

2.1.4 Human Sexuality

Sexuality is an integral part of a human life and wellbeing. Sexuality in health and illness includes a wide spectrum of excitations and activities, which are a source of different drives, affects impulses like pleasure, anxiety, feelings of shame, disgust, etc., throughout the lifespan

[75]. Proper sexual functioning is one of the most important components of quality of life and of maintaining a satisfying intimate relationship [23]. When sexual health encounters a problem and if not well addressed, it can lead to psychological trauma, frustration and may even heighten the already existing problem thus lowering the self-esteem. A healthy sexuality requires balance of physical, psychological and social factors and an imbalance in one or more of these factors can result in a sexual dysfunction [24].

2.1.5 Assessment of erectile dysfunction

History and physical examination are the main assessment tools for successful diagnosis of ED. ED questionnaires serve as important adjunct to the proper diagnosis of ED. The patient fills out the questionnaire to delineate the extent of the erection problem

Many male sexual function profiles and ED questionnaires have been developed. The International Index of Erectile Function Questionnaire (IIEF) has been reported to be the most reliable measure of ED [3, 12]. It has 15 questions and the total score is obtained by the sum of the individual scores of each question. It addresses and quantifies five domains: erection function, orgasmic (ejaculation) function, sexual desire (libido), intercourse satisfaction (ability to sustain intercourse), and overall satisfaction/premature ejaculation domains. The questionnaire is appended in this paper.

Two other questionnaires have also been used in clinical studies- The Sexual Encounter Profile (SEP) and the Global Assessment Questionnaire (GAQ) [12]. These questinnaires are superseded by the IIEF.

As part of assessment, some focused laboratory and non laboratory measures can be done to look for factors associated with ED. Laboratory tests may include: Lipid profile, Glycosylated haemoglobin and Fasting blood glucose. Non-Laboratory tests include: blood pressure, weight and height.

2.1.6 Treatment of erectile dysfunction in general

Initially, preventive measures will help reduce the risk of developing ED. Improving glycemic control and hypertension, ceasing cigarette smoking, increased level of regular physical activity and reducing excessive alcohol intake has been shown to benefit patients with ED. Avoiding or substituting medications that may contribute to ED is also helpful [76, 77].

Once ED has developed, oral agents to improve sexual function may be considered. Sildenafil (Viagra) acts by blocking the catabolism of cGMP, resulting in an increase in nitric oxide. Fifty-six percent of diabetic men with ED experience improvement of erectile function with sildenafil, compared to 70% of nondiabetic men with ED [77]. Phosphodiaesterase type 5 (PDE5) inhibitors are contraindicated in those who are on nitrates, because of the potential for a dramatic fall in blood pressure.

Another oral treatment that has been used with very little success is yohimbine (Yocon, Yohimex). This is an alpha 2 adrenergic receptor blocker that increases cholinergic and decreases adrenergic tone. It stimulates the mid-brain and increases libido.

For those patients who are not candidates for oral therapy, intracavernosal injections are an acceptable alternative. Injections with alprostadil (Caverject) and papaverine (Genabid) have been used to induce erection. This form of therapy has a response rate of well over 70% [78].

Mechanical therapy is also effective and is especially well-accepted in men with stable partners. Vacuum-assisted erection devices are effective in creating erections in as much as 67% of cases. Penile prosthesis is a viable option for men who cannot use sildenafil and who find the injections or vacuum erection therapy distasteful [77, 79] .

Testosterone therapy with injections or patches should be tried in patients with documented low testosterone levels [80].

Psychotherapy should be offered to the patients and their partners to address any interpersonal conflicts, because ED is a problem for couples—not just men [77, 81].

The application of gene therapy for ED represents an exciting new field. Although preclinical studies have highlighted the application of local gene therapy as a viable treatment option for ED in diverse pathologic conditions including diabetes, ageing, hypercholesterolaemia and cavernous nerve injury, this therapeutic approach still requires more clinical studies in humans [82, 83]

Vacuum, Local injection and prosthesis may pose problems to our local patient due to taboo and culture as well as partner acceptance. Moreover, they may pose risk of infections to diabetic patient especially those with poor hygiene.

2.1.7 Prevention of ED

The mainstay for prevention of ED includes correcting the modifiable risk factors for atherosclerotic vascular disease. Glycemic control should be optimized with oral anti-diabetic drugs or insulin while the blood pressure and lipid levels should be normalized with appropriate medications.

Patient should be encouraged to quit smoking, to reduce their alcohol intake and to give up recreational drugs.

Prescription drugs and over the-counter medications should be checked for possible contribution to ED. Effort should be made to avoid or to find substitutes for drugs with negative impact of sexual function [84].

Relationship counseling and psychiatric medications is useful to treat anxiety and depression [85].

2.2 PROBLEM STATEMENT

ED is an under-recognized, under-discussed, and commonly untreated complication of diabetes [86]. But it is also one of the most preventable and treatable diabetic complications. It is a "couple's disorder," affecting both the patient and his partner. ED constitutes a major public health problem, influencing the patient's well being and quality of life. It also leads to broken homes and marital disharmony , psychological, social, and physical morbidity [34].

Erectile dysfunction in diabetic men is often undiagnosed unless the healthcare provider specifically asks the patient about possible symptoms. Obviously this is a conversation that many patients feel uncomfortable about initiating due to taboo/stigma and because it touches somebody's personal life. At present, Tanzania has no guidelines to ensure that men with diabetes are asked for ED at clinic visits.

Knowledge of erectile dysfunction is rapidly expanding, and effective new treatments are now available, including oral medications, injectable drugs, vacuum devices, and inflatable prostheses. It is therefore important for both physicians and patients to be educated and made aware of the causes and treatments of ED.

Despite advances in understanding of erectile dysfunction, its risk factors and the possible treatment; little is known in Tanzania on the prevalence, pattern and associated factors among Tanzanian diabetic men.

Of the few studies on ED which have been done in Tanzania, none have included other sexual function domains. In order to treat ED effectively one need to determine which domains of sexual dysfunction is mostly affected.

Moreover, risk factors associated with ED in Tanzanian diabetic men may be different to those in other population. Understanding the associated factors will help in establishing appropriate preventive measures.

2.3 RATIONALE

ED is one of the most important complications of DM; it is also associated with cardiovascular disease such as hypertension. Studies show that some determinant factors of ED among diabetic men are modifiable e.g. Poor glycemic control, hypertension, use of medications e.g. antihypertensives, and cigarette smoking. Few studies in Sub-Saharan Africa has been done and published regarding ED among diabetic men, but none in our country.

This study was conducted to determine the magnitude and severity of ED in diabetic men, the pattern (domain) of ED, and the associated risk factors.

It was therefore justifiable to have this study, the outcome of this study will provide an insight on how to screen and address the modifiable risk factors for ED and improving lifestyles of this group of patients including ED treatment. This study is unique because it relates ED with other sexual dysfunction domains.

The study findings will also be the basis for future research among this population.

The IIEF has been linguistically validated in 32 languages excluding Swahili language, in this study we translated the IIEF into Swahili language at the Institute of Kiswahili Studies, University of Dar es Salaam, and piloted before it was administered to the study.This Swahili translated tool can be widely used in Swahili speaking countries to assess ED among patients and for other research purposes.

2.4 OBJECTIVES

2.4.1 Broad Objective

To determine the prevalence of erectile dysfunction and associated factors among diabetic men attending the diabetic clinic at Muhimbili National Hospital (MNH), Dar-es-Salaam.

2.4.2 Specific objectives

1. To determine the prevalence of erectile dysfunction among male diabetic patients attending diabetic clinic at MNH, Dar-es-Salaam.
2. To determine the severity of erectile dysfunction among male diabetic patients attending diabetic clinic at MNH, Dar-es-Salaam.
3. To determine the pattern of sexual function domains among male diabetic patients attending diabetic clinic at MNH, Dar-es-Salaam
4. To determine factors influencing erectile dysfunction among male diabetic patients attending diabetic clinic at MNH, Dar-es-Salaam.
5. To describe the association between erectile dysfunction and other sexual function domains among male diabetic patients attending diabetic clinic at MNH, Dar es Salaam.

CHAPTER THREE

3.0 METHODOLOGY

3.1 Study design

This study was a hospital-based descriptive cross-sectional.

3.2 Study area

The study was conducted at the Diabetic Clinic, Muhimbili National Hospital (MNH) in Dar es Salaam. MNH is a national referral, consultant and teaching hospital located in Dar es Salaam city.

Dar es salaam is the largest commercial city in Tanzania, had a population of 2.5 million people in 2002, currently estimated to have over 4 million residents and is expected to reach 5.12 million by 2020 [87]. The city is administratively divided into three districts, namely Kinondoni, Temeke and Ilala.

MNH diabetic clinic is a well established clinic which offers care to diabetic patients who are either referred from other diabetic centers in Dar es Salaam or from other hospital in the nearby regions due to management difficulties

The clinic provide broad services including assessment of diabetic patients for anthropometric measurements, fasting blood glucose, glycosylated hemoglobin, urine dipstick and other relevant quick assessment depending on patient presentation. The clinic also offers for free the oral hypoglycemic and insulin (with syringes).

The clinic has adequate space to carry out its services; there are three separate rooms which are sufficient for providing privacy between patients and the doctor.

About six doctors and 4 nurses are present at the day of clinic. There are two days of clinic per week, on Monday and Thursday. The Monday session is mainly for paediatric diabetic patients. This study included only patients attending the Thursday clinic.

3.3 Study population and participants

The study population was diabetic men attending diabetic clinic, Muhimbili National Hospital. All booked and newly registered patients attending the diabetic clinic who satisfied inclusion criteria were eligible for the study.

3.4 Inclusion criteria

The study participants included diabetic male patients attending diabetic clinic MNH, with the following criteria

- Age ≥ 18 years
- Duration of diabetic ≥ 1 year
- Both type 1and 2 diabetic mellitus
- Consenting to participate in the study

3.5 Exclusion criteria

- The very sick patients for purpose of this study was defined as: Patients with unstable vital signs/mental status e.g in diabetic ketoacidosis or hyperosmolar hyperglycaemic state, confused, or in septicaemia as in infected diabetic foot.

3.6 Sample size calculation

Sample size was calculated from the formula for cross-sectional studies.

$$N = \frac{z^2 p q}{\varepsilon^2}$$

Whereas:

N =sample size

z= point on standard normal distribution curve such that the area to the right of it is 2.5% (z=1.96)

p= estimated prevalence of erectile dysfunction (among diabetic Kenyan men) = 74.1%

q= 100-p = 25.9

Ɛ= margin of error on p (set at 5%)

$N = \frac{1.96^2 \times 74.1 \times 25.9}{5 \times 5} = 294$

N = 294

Allowing for 5% non-response, adjusted minimum sample size was N' ≈ 310.

3.7 Sampling technique

The diabetic clinic was visited once a week on a Thursday. The average number of patient per clinic visit was about 60 of whom male clients were about one third i.e the clinic is relatively predominated by females.

Study participants were recruited consecutively. The target was to recruit 10 patients per day. On the clinic day the first ten male patients to attend were identified, given numbers and recruited, those who met the inclusion criteria. The first come first saved policy was employed. If the patient did not consent then the next male in the list was recruited, until the required number of 10 patients per clinic day was reached. Patient's files were labeled after recruitment to avoid repetition.

3.8 Study Period

The study period was from May to December 2011

3.9 Data Collection Technique (Procedures)

The participants were interviewed using a standard structured questionnaire which included socio-demographic data, to assess the different sexual domains; the International Index of Erectile Function (IIEF) Questionnaire was used. The IIEF was translated into Kiswahili language and the author asked all the 15 questions.

3.9.1 The IIEF Questionnaire

Refer to section 2.1.5

3.9.2 Laboratory tests

The blood specimens for laboratory investigations {lipid profile, Fasting or random blood glucose, and Glycosylated haemoglobin (HbA1c)} were collected in vaccutainer tubes for analysis at MOI's main laboratory. Cobas Integra 400 plus (Roche) clinical chemistry analyser was used for lipid profile analysis, Accu-check for FBG, and DCA 2000+ (Bayer) for HbA1c analysis. The sample was sent to laboratory for analysis within 30mins after collection.

3.9.3 Anthropometric measurements

Weight: The individual's body weight was measured by standardized (AXIOM, AX – 120) scales without heavy clothing and shoes, and recorded to the nearest 0.5kg

Height: The study participant's height was measured with a firm measure against a vertical wall, and the height was measured to the nearest 0.5cm

The body mass index (BMI) was calculated using the formula Kg/m^2; where Kg is body weight in kilograms and m is squared height in metres. The BMI will be classified according to World Health Organization [88] into underweight: BMI< 18.5; Normal weight: BMI 18.5-24.9; overweight: BMI 25.0-29.9; obesity: BMI>30

The research assistants were trained on how to take the anthropometric measurements prior to the commencement of data collection.

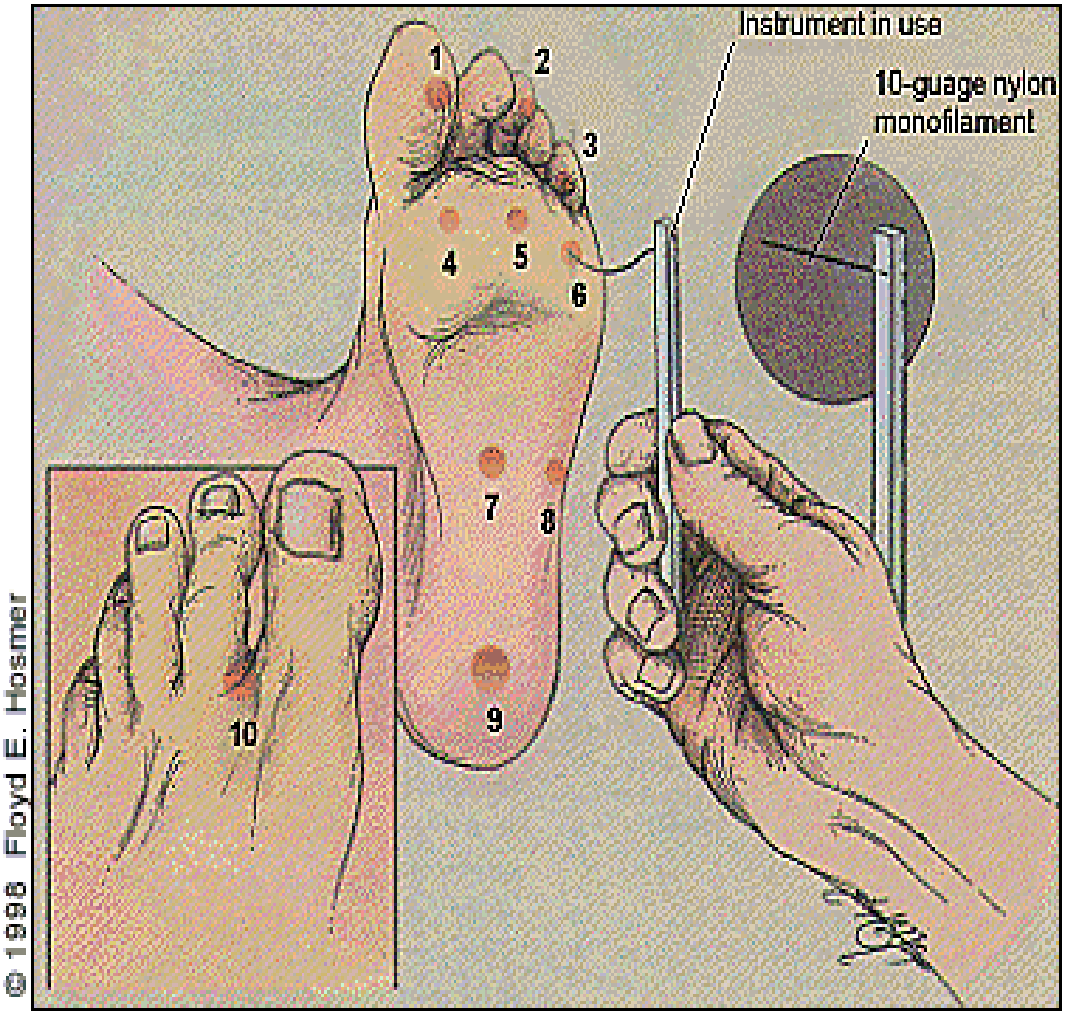
3.9.4 Blood pressure (BP) measurements

The patients' blood pressure was measured by trained research assistants. After a 10-minutes rest, the patients BP was measured with a standard manual mercury sphygmomanometer while in the sitting position. The systolic blood pressure (SBP) and diastolic blood pressure (DBP) measurements was determined by the 1st and 5th Korotokoffs sounds respectively. The BP was measured twice, with interval of 5 minutes; the average of the two BP measurements was considered as the patient's BP. Hypertension was defined as SBP≥140mmHg and/or DBP≥90mmHg [89] or known hypertensive on treatment.

3.9.5 Screening test for peripheral neuropathy

The 10g Semmes Weinstein monofilament (NovoMix 30) was used to screen diabetic patients for peripheral neuropathy. Patients was asked to close their eyes while the monofilament is pressed perpendicular 9 areas of the plantar surface and one on dorsal surface of the feet (Fig 1) until it buckles. The patients were asked to say 'Yes' each time they feels the filament. Failure to feel the filament at 4 of 10 sites is 97% sensitive and 83% specific for identifying loss of protective sensation[90].

Fig 1. The nylon monofilament test



3.9.6 Evaluation for peripheral vascular disease

Ankle Brachial Index (ABI), the ratio of SBP of the brachial artery to that of pedal artery was measured to assess lower limb blood flow.

A bedside, Hand-held Doppler ultrasound machine (MEDA SONICS), 8 MHz probe was used, Ultrasound transducer gel, Sphygmomanometer and cuff, and a Calculator.

Patients were provided with information about the procedure to relieve any anxieties they may have. The room temperature was ensured comfortable. Patients were asked to remove any tight articles of clothing, which may cause pressure on the blood vessels proximal to the site where the blood pressure is being measured. The patient was asked to lie flat and rest for a minimum of 10 minutes to obtain a resting pressure.

Measurement of the brachial systolic blood pressure: A sphygmomanometer cuff of an appropriate size was selected and wrapped around the patient's upper arm just above the elbow, the brachial pulse was palpated and ultrasound gel applied; the Doppler probe was angled at 45 degrees to the direction of the blood flow (towards the heart) and the position was adjusted to locate the best signal; the sphygmomanometer cuff was inflated until the signal disappears, then the cuff was slowly deflated and the pressure recorded at which the signal returns.

Measuring the ankle systolic pressure: An appropriately sized sphygmomanometer cuff was selected and placed around the leg just above the ankle; the posterior tibial artery was palpated, ultrasound gel was applied and the best signal was located. The sphygmomanometer cuff was inflated until the signal disappears, then the cuff was slowly deflated and the pressure recorded at which the signal returns.

ABPI = highest ankle systolic pressure/highest brachial systolic pressure

< 0.9 abnormal (at risk of ulceration)

0.9 to 1.0 normal

> 1.3 non-compressible: The result may be falsely elevated in patients with diabetes due to calcification of the medial layer of the artery, preventing the arteries from being compressed

An ankle brachial pressure index (ABPI) is a simple non-invasive method of identifying arterial insufficiency within a limb. It compares the ankle and brachial systolic blood pressures. An important factor determining the rate of healing of any wound is adequate

arterial blood supply. In the management of leg ulcers, the ABPI forms a fundamental part of the assessment

In the past, a clinical examination would have involved palpating the pulses to detect any arterial impairment. However, the evidence is that palpation of pulses is subjective and prone to considerable error[91].

Accurate assessment is necessary to determine the correct aetiology of the ulcer and exclude those patients with arterial disease for whom compression is dangerous[92].

3.9.7 Laboratory procedure

Biochemical tests: Fasting blood samples for total serum cholesterol, high density lipoproteins (good cholesterol), low density lipoprotein (bad cholesterol), serum triglycerides and blood glucose, estimation was taken. Five millilitres of venous blood was taken from the antecubital fossa and placed in empty sterile tubes. The samples were taken to Muhimbili Orthopedic Institute (MOI) laboratory for analysis within a day. MOI laboratory was chosen because the study investigator got an assistance of doing these test free of charge . Quality assurance prior to carrying out sample processing and analysis was adhered to, these were done by a trained laboratory technician who was also given proper information of this study.

Blood samples for Glycosylated haemoglobin was collected and analyzed at the diabetic clinic, using DCA 2000+ (Bayer). Due to financial constraints, glycosylated haemoglobin was only done in 145 patients. These were selected on alternating basis with aim of getting 5 patients per day, so from the 10 patients recruited per day the 1st one was selected for HbA1c then the 3rd, 5th etc. Patients with HbA1c done within 6 months were not re-checked and their reading was used in this study.

3.10 Data processing and analysis

The collected data were checked for quality and coding was done prior to entering into the computer (SPSS ver. 15.0) statistical program. Analysis was done using statistical package for social sciences (SPSS) version 15.0. The descriptive statistics were computed, comparisons of

variables were done with Chi-square test for categorical variables. Any variable which was significant on Univariate analysis was subjected to Multivariate logistic regression analysis to determine predictors of erectile dysfunction. P value of < 0.05 was considered significant to provide evidence of significant differences or associations.

3.11 Ethical consideration

Ethical clearance was sought from the Chairperson, Research and Publications Committee of Muhimbili University of Health and Allied Sciences (MUHAS).The permission was sought from authorities of Muhimbili National Hospital diabetic clinic. The information about the study was given to patients prior the interview and samples collection. A written consent was obtained from each patient. The confidentiality was ensured to all individuals who participated in the study.

3.12 Patients disposal

Patients who were found to have erectile dysfunction were counseled (those who came as a couple were counseled as thus after obtaining consent), about the available treatment options and their advantages/side effects. Prescriptions were written for those who agreed to be put on treatments, confidentiality was maintained. Patients with evidence of peripheral vascular disease were planned to regularly attend the clinic every 2months for check-up. Those with hyperlipidaemia was prescribed lipid lowering drugs and the newly diagnosed hypertensive were managed accordingly i.e lifestyle changes+/- antihypertensives. Lipid profile results and HbA1c results were put in patient's file for being seen by physician in next clinic visits.

CHAPTER FOUR

4.0 RESULTS

4.1 Sociodemographic characteristics of male diabetic patients attending Diabetic clinic at MNH

Over a period of 8 months, May to December 2011, 503 diabetic male patients attended at MNH diabetic clinic, 312 (62%) were recruited in this study. Most, 256 (82.1%) of the participants were married (Table 1).

There were 90 (28.8%) patients in the young age group (below 45 yrs) while majority 222(72.2%) were above 45 years, overall mean age was 51.33 (SD=15.03) years. Majority, 152(48.7%) of participants attained primary school education and 114(36.5%) were self employed (Table 1).

Two hundred and fifty one (80.4%) patients were T2DM and sixty one (19.6%) patients were T1DM. The mean diabetic duration of this study population was 8.99 (SD = 7.62) years with a minimum of 1year and maximum of 37years.

Among these patients, the mean fasting blood glucose was 10.38 (SD = 0.78) mmol/l. The mean Glycosylated haemoglobin (HbA1c) was 8.98 (SD = 2.37) %, in 145 patients tested for HbA1c.

Table1. Sociodemographic factors of male diabetic patients attending Diabetic clinic at MNH (N=312)

Characteristics	Frequency (N)	%
Age group (yrs)		
< 30	35	11.2
30 – 44	55	17.6
45 - 59	113	36.2
60+	109	34.9
Marital status		
Married	256	82.1
Single	36	11.5
Divorced	4	1.3
Cohabiting	7	2.2
Widowed	9	2.9
Employment		
Peasant	71	22.8
Government	38	12.2
Private sector	57	18.3
Self employee	114	36.5
Others	32	10.2
Level of education		
Informal	40	12.8
Primary	152	48.7
Secondary	82	26.3
Post secondary	38	12.2

With respect to diabetic medications use, 225 (72.1%) of patients were on oral hypoglycemic agents (OHA), 76 (24.1%) on insulin, and 11(3.5%) were on other medications/treatment options.

Other medications/options include diet, herbs, Chinese medications. One patient was on insulin and herbs, one on both OHA and herbs, and two on both OHA and insulin. Among patients on OHA, 85 (37.8%) were on a single OHA, 131 (58.2%) on two OHA, and 9 (4%) on three OHA.

4.2 Prevalence and severity of erectile dysfunction and other sexual domains of the male diabetic patients attending diabetic clinic at MNH

The prevalence of erectile dysfunction was found to be 172 (55.1%). Forty patients (12.8%) had mild dysfunction, 45 (11.5%) had moderate dysfunction, and 87 (27.9) had severe dysfunction (Table 2)

The prevalence of dysfunction for other sexual function domains were 62.5%, 54.2%, 49.0%, and 59.9% for sexual desire, orgasmic function, intercourse satisfaction, and overall satisfaction respectively. Prevalence of ED seems to be similar to prevalence of other sexual dysfunction domains (range 49.0% to 62.5%). Severity of all dysfunctions is also similar, and tends to increase with increasing dysfunction (Table 2)

Table 2. Level of dysfunction by type of sexual function domains among male patients attending the diabetic clinic at MNH (N=312)

Level of dysfunction	Type of sexual function domain				
	Erectile function, n (%)	Sexual desire, n (%)	Orgasmic function,n (%)	Intercourse satisfaction,n (%)	Overall satisfaction, n (%)
None	140 (44.9)	117 (37.5)	143 (45.8)	159 (51)	125 (40.1)
Mild dysfunction	40 (12.8)	61 (19.6)	43 (13.8)	33 (10.6)	54 (17.3)
Moderat dysfunction	45 (11.5)	46 (14.7)	38 (12.2)	34 (10.9)	45 (14.4)
Severe dysfunction	87 (27.6)	88 (28.2)	88 (28.2)	86 (27.6)	88 (28.2)
Overall dysfunction	**172(55.1)**	**195(62.5)**	**169(54.2)**	**153(49.0)**	**187(59.9)**

When severity of ED was classified by age group, it was found that the prevalence of ED increases with increasing age, the trend is particularly observed on the severe form of ED. The observed difference was statistically significant (p<0.001) - Table 3

Table 3. Severity of ED by age group among male patients attending the diabetic clinic at MNH (N=312)

Age group (yrs)	Erectile dysfunction severity*				Total, N(%)
	No dysfunction, N (%)	Mild dysfunction, N (%)	Moderate dysfunction, N(%)	Severe dysfunction, N(%)	
< 30	31 (88.5)	1 (2.9)	1 (2.9)	2 (5.7)	35 (100)
30 - 44	35 (63.7)	7 (12.6)	5 (9.1)	8(14.6)	55 (100)
45 - 59	52 (46.0)	12 (10.6)	21 (18.6)	28 (24.8)	113 (100)
60+	22 (20.2)	20 (18.3)	18 (16.5)	49 (45)	109 (100)

*p<0.001 by X^2 test

4.3 Sociodemographic and clinical factors associated with ED among male diabetic patients attending diabetic clinic at MNH

Table 4 shows sociodemographic factors associated with ED in this study group, it was found that erectile dysfunction increases significantly with age. The prevalence was 78% in men aged 60 years and above (p < 0.001), there is a trend towards increase in prevalence of erectile dysfunction as the age increases.

The prevalence of ED was higher in patients who did not attain formal education (70%) than those in other levels of education; the difference was not statistically significant, p=0.16.

ED was higher among unemployed individuals (71.8%) than among employed individuals; the difference was statistically significant, p= 0.03.

The prevalence of ED was significantly higher in patient who presented with longer duration of diabetes of more than 10yrs (68.2%) than those who presented with the duration (less than 10yrs (48%); p<0.001.

In this study, prevalence of erectile dysfunction was significantly higher in T2DM (62.5%) than in T1DM (23%); p<0.001.

Other risk factors which were highly statistically significant associated with high prevalence of ED in this study group included history of cigarette smoking, history of drinking alcohol, past history of stroke, history of peripheral neuropathy and being hypertensive (p<0.05).Table 4.

Table 4. Univariate analysis of sociodemographic and clinical factors associated with ED among male diabetic patients attending diabetic clinic at MNH (N=312)

Variable	Number	Prevalence (%) of ED	p-value [a]
Age group (yrs)			
< 30	35	6 (17.1)	<0.001*
30– 44	55	23 (41.8)	
45 – 59	113	58 (51.3)	
60+	109	85 (78.0)	
Education level			
None	40	28 (70)	0.16
Primary	152	82 (53.9)	
Secondary	82	40 (48.8)	
Post secondary	38	22 (57.9)	
Current employment			
Peasant	71	51 (71.8)	0.03*
Government	38	19 (50.0)	
Private	57	28 (49.1)	
Self employment	114	58 (50.9)	
Others	32	16 (50.0)	
Duration of diabetes (years)			
< 10	180	83 (46.1)	0.001*
10 – 19	106	70 (66.0)	
> 20	26	19 (73.1)	
Type of diabetes			
T1DM	61	14 (23.0)	<0.001*
T2DM	251	158 (62.9)	
H/o smoking			
Never smoked	252	131 (52)	0.02*
Ever smoked	60	41 (68.3)	
H/o alcohol			
Never drunk	182	92 (50.5)	0.05*
Ever drunk	130	80 (61.5)	
Hypertensive**			
No	154	68 (44.2)	<0.001*
Yes	158	104 (65.8)	
H/o stroke			
No	291	155 (53.3)	0.01*
Yes	21	17 (81.0)	
H/o p/neuropathy			
No	146	59 (40.4)	<0.001*
Yes	166	113 (68.1)	

[a] X^2 test,*p<0.05, **20 patients were found to have ↑BP and thus classified as hypertensive

4.4 Clinical parameters associated with ED among male diabetic patients attending diabetic clinic at MNH

Table 5 shows the clinical parameters associated with ED in this study popultion. Patient who had low (4 out of 10) monofilament score were highly statisticaly significant had ED as compared to those who had normal score, 81.1% Vs 43.8% ($p<0.001$).

The prevalence of ED was high among patients with high level of total cholesterol (>5.2mmo/l), overweight (BMI = 25 – 29.9), high fasting blood sugar (>7.1mmol/l), although the observed difference was not statistically significant ($p>0.05$). Also the prevalence of ED was high in patients with normal levels of HDL-c (0.9-2.00mmol/l), LDL-c (≤3.34mmol/l), Triglcerides (≤2.3mmol/l), and HbA1c (≤7%) but also the observed difference was not statsticaly significant ($p>0.05$).

Table 5 also shows that the prevalence of ED among patient with evidence of peripheral vascular disease (PVD) as revealed by abnormal ankle brachial index score {<0.9 (41 patients), and > 1.3 (7 patients)} is highly statisticaly significant high (93.8%) than those without PVD (48.1%) $p<0.001$.

Table 5. Univariate analysis of clinical parameters associated with ED among male diabetic patients attending diabetic clinic at MNH (N=312)

Variable	Number	Prevalence (%) of ED	p-value [a]
Monofilament test			
Could feel	217	95 (43.8)	<0.001*
Could not feel	95	77 (81.1)	
LDL-c (mmol/l)			
Normal (≥3.34)	210	118 (56.2)	0.59
Abnormal (<3.34)	102	54 (52.9)	
Triglyc (mmol/l)			
Normal (≤ 2.3)	266	149 (56.0)	0.45
Abnormal (>2.3)	46	23 (50.0)	
Total chol (mmol/l)			
Normal (≤ 5.2)	209	113 (54.1)	0.59
Abnormal	103	59 (57.3)	
Fasting b/sugar			
Normal (≤ 7)	103	55 (53.4)	0.66
Abnormal (>7)	209	117 (56.0)	
HbA1c (%) [b]			
Normal (≤ 7%)	32	20 (62.5)	0.49
Abnormal (7%)	113	63 (55.8)	
Ankle Brachial Index score			
Normal	264	127 (48.1)	<0.001*
Abnormal	48	45 (93.8)	
Bodymass index[c]			
<18.5	20	9 (45)	0.27
18.5 – 24.9	152	77 (50.3)	
25 – 29.9	90	56 (62.2)	
>30	38	20 (52.6)	

Note : [a]Chi square test, *p<0.05

[b]HbA1c was done among 145 patients

[c]Among the twelve individuals, body mass index could not be determined because these patients each have one limb amputated and were not stable to stand for weight and height determination.

4.5 Predictors of ED among male diabetic patients attending diabetic clinic at MNH

Any factor with p-value <0.05 on univariate analysis were considered for multivariate analysis so as to control for confounder risk factors. Table 6 shows factors associated with ED on multivariate logistic regression analysis; old age (more than 60years), evidence of peripheral neuropathy on monofilament test, and evidence of peripheral vascular disease were predictors of erectile dysfunction. Other factors could not maintain statistical significance multivariate logistic regression.

It was revealed that ED is more likely to occur seven times in patients aged more than 60 years (OR=7.074, 95% CI=1.229-40.688) as compared with men 29 years and younger (Table 6). Also, patients who could't feel on monofilament test are two times more likely to have ED (OR=5.858, 95% CI=1.613-21.276) than those who could feel. Patients with evidence of peripheral vascular disease are five times more likely to have ED (OR=2.464, 95% CI 1.153-5.262) than those without evidence of PVD.

A very wide confidence interval on age and monofilament test parameters could reflect lack of precision.

Table 6. Multivariate analysis of factors associated with ED among male diabetic patients attending diabetic clinic at MNH (N=312)

Variable *	aOR	95% CI	p-value [a]
Age group (yrs)			
< 30	1		
30– 44	3.493	0.714 – 17.082	0.122
45 – 59	3.533	0.635 – 19.669	0.149
60+	7.074	1.229 – 40.688	0.028
Duration of diabetes (years)			
< 10	1		
10 – 19	1.750	0.914 – 3.353	0.092
> 20	1.481	0.491 – 4.463	0.485
Type of diabetes			
T1DM	1		
T2DM	2.695	0.947 – 7.666	0.063
H/o smoking			
Never smoked	1		
Ever smoked	1.346	0.652 – 2.780	0.421
H/o alcohol			
Never drunk	1		
Ever drunk	1.266	0.694 – 2.308	0.442
H/o stroke			
No	1		
Yes	2.628	0.771 – 8.962	0.122
H/o p/neuropathy			
No	1		
Yes	1.464	0.846 – 2.665	0.211
Hypertensive			
No	1		
Yes	1.139	0.610 – 21.276	0.683
Monofilament test			
Could feel	1		
Couldn't feel	2.464	1.153 – 5.267	0.019
ABI score			
Normal	1		
Abnormal	5.858	1.613 – 21.276	0.007

[a]Chi square test, *the reference is first group in each category, aOR = adjusted odds ratio ABI=Ankle Brachial Index

4.6 Hypertension and ED among male diabetic patients Patients who were known to be hypertensive had a significantly high prevalence of of ED, 95/138 (68%) compared to those those not known to be hypertensive 77/174 (44.3%) p <0.001. It is known fact that hyperetnsion is associated with other factors which can also have an additive effect on ED like insulin resistance and hyperlipidaemia or even the side effect of drugs used to treat hyertension.

Table 7 shows the association between ED with the current use of antihypertensive, it was found that the use of Angiotensin II receptor blockers (ARB) was significantly associated with ED with the prevalence (74.6%) of ED (p<0.05) as compared to other antihypertensive drugs. All 138 patients known to have hypertension were using antihypertensive, some with a combination of more than one antihypertensive.

Table 7. Association between current antihypertensive drugs (for patients known to be hypertensive) and ED among male diabetic patients attending diabetic clinic at MNH (n=138)

Drug	Total	Prevalence (%) ED	p-value [a]
Beta blockers			
No	113	81 (71.7)	0.126
Yes	25	14 (56.0)	
ACEIs			
No	85	63 (74.1)	0.09
Yes	53	32 (60.4)	
ARBs			
No	66	40 (60.6)	0.046*
Yes	72	55 (76.4)	
Diuretics			
No	122	84 (68.9)	1.00
Yes	16	11 (68.8)	
CCBs			
No	105	74 (70.5)	0.46
Yes	33	21 (63.6)	

Note : [a]Chi square test , *p< 0.05, ACEIs=Angiotensin Converting Enzymes Inhibitors, ARBs= Angiotensin Receptor blockers, CCBs= Calcium Channel Blockers

4.8 Association between erectile dysfunction and other sexual function domains among male diabetic patients attending diabetic clinic at MNH

Table 8 shows that Erectile dysfunction was statistically significant highly associated with other sexual domains, whereby 84.6% of patients with ED had reduced libido, 97.0% had ejaculation disorders, 98.7 had intercourse dissatisfication, and 88.2% had overall intercourse dissatisfaction. All of the above findings show in patients with ED, that the possibilities of having normal sexual desire, normal orgasmic functions, intercourse satisfaction or overall sexual satisfaction is almost zero.($p<0001$).

Table 8. Association between Erectile dysfunction and abnormality in other Sexual function domains among male diabetic patients attending diabetic clinic at MNH (N=312)

Sexual function domain	Status of sexual function domain	Total	Prevalence (%) of ED	p-value [a]
Sexual desire	Normal	117	7 (6.0)	< 0.001*
	Abnormal[b]	195	165 (84.6)	
Orgasmic function	Normal	143	8 (5.6)	< 0.001*
	Abnormal	169	164 (97.0)	
Intercourse satisfaction	Normal	159	21 (13.2)	< 0.001*
	Abnormal	153	151 (98.7)	
Overall satisfaction	Normal	125	7 (5.6)	< 0.001*
	Abnormal	187	165 (88.2)	

Note : [a]Chi square test, *$p<0.05$, [b]Abnormal comprises mild to severe dysfunction

4.9 Onset of ED, use of medications for ED and outcome of use of medications for ED among male diabetic patients attending diabetic clinic at MNH

In this study, 84.9% of patient had ED for more than 1 year, onset of ED was arbitrary taken as the first day the patients noted having ED as per definition of ED. Only 24(14%) had used medication to improve erection of which 91.7% was oral medications. Half of patients who used medications got satisfied (Table 9).

Table 9. Onset of ED and use of medications for ED among male diabetic patients attending diabetic clinic at MNH (n=172)

Variable	Frequency	%
Onset of ED (months)		
Within 3	2	1.2
3 to 12	24	13.9
>12	146	84.9
Ever tried Medications		
No	148	86
Yes	24	14
Oral medications (**PDEi**)		
No	2	8.3
Yes	22	91.7
Other medications		
No	22	91.7
Yes	2	8.3
Satisfied with medications		
No	12	50
Yes	12	50

PDEi= Phosphodiasterase inhibitors

CHAPTER FIVE

5.0 DISCUSSION

5.1 Prevalence and severity of erectile dysfunction among diabetic male patients attending diabetic clinic at MNH

The present study aimed at determining the prevalence of ED and associated risk factors among diabetic male patients attending diabetic clinic at MNH.

This study included type 1 and type 2 diabetic male patients with mean age of 51.3 +/-15.0 years, mean diabetes duration 8.9+/-7.6years, and mean HbA1c 8.9+/-2.4%. By the use of the IIEF and direct questioning, the prevalence of erectile dysfunction was found to be 55.1% (n= 172). Forty patients (12.8%) had mild dysfunction, 45 (11.5%) had moderate dysfunction, and 87 (27.9%) had severe dysfunction.

The prevalence was higher (78%) in men aged 60 years and above ($p < 0.001$); there was a trend towards increase in prevalence of erectile dysfunction as the age increases. Longer duration of DM, type 2 DM, history of peripheral neuropathy, history of hypertension, low score on monofilament test, and evidence of peripheral vascular disease were highly significantly associated with ED ($p =<0.001$). History of smoking, history of drinking alcohol, and history of stroke were also significantly associated with ED ($P = 0.02$, 0.05, and 0.01 respectively). On multivariate logistic regression the predictors for ED were found to be old age (odds ratio [OR] = 7.1, 95% CI 1.2-40.7), evidence of peripheral neuropathy ([OR] =5.9, 95% CI 1.6-21.3), and evidence of peripheral vascular disease ([OR] =2.5, 95% CI 1.2-5.3) {p= 0.028, 0.019, and 0.007 respectively}. This study also showed that ED was highly statistically significant associated with other sexual domains ($p <0.001$).

Erectile dysfunction is a distressing complication of diabetes. The prevalence of ED among diabetic men varies from 35 to 90% [6, 53, 54, 63, 72]. In the Massachussetts male aging study, ED was three times more common in diabetic compared with nondiabetic men[5].

The observed prevalence from the current study is lower than that observed from studies done in Asia whereby in Hong Kong, Siu et al[6] who recruited 500 type 1 and type 2 diabetic male

patients with mean age 58.3+/-10years, mean diabetes duration of 5years, and mean HbA1c 7.7+/-1.5% reported the prevalence of 63.2% by direct questioning by interviewer while in S.Korea, Cho et al[53], studied 1,312 type 1 and type 2 diabetic patients with mean age 53.8+/-6.6years, mean duration of DM of 6years, and mean HbA1c 7.93% and found the prevalence of ED to be 65.4% by telephone and face-to-face interview.

On the other hand, Khatib F.A et al [70] reported the overall prevalence of ED among 988 married Jordanian men with diabetes to be 62%, they found that 30.3% had severe ED. The study was conducted from January to August 2004.

In Sri Lanka, Malavige et al[54] who studied 253 type 2 DM patients with mean age 55.6+/-10.4 years, mean DM duration 7.2 years, and mean HbA1c 7.2+/-1.6% gave a prevalence of 73.1% by the use of IIEF5.

Some studies done in Europe revealed low prevalence of ED as compared to the current study. In the UK, McCulloch et al[63] conducted a study among 541 type 1 and type 2 diabetic male patients aged 20 to 69 years by using a self-reported nonvalidated questionnaire and found a prevalence of ED to be 35%, while in Italy, Fedele et al[72] studied 9,868 type 1 and type 2 diabetic male patients by direct questioning by interviewers and found a prevalence of ED of 36%.

Among prevalent studies done in Afirican diabetic male, the prevalence observed from the current study is lower from that of the study done at Kenyatta National Hospital(KNH) by Ngalyuka et al[74] from Oct 2007 to March 2008 among T2DM patients attending the KNH diabetic clinic , ED was found to be five times more prevalent in diabetic males compared to age-matched controls with a prevalence of 74.3% and 15.1 % in diabetics and controls respectively, the IIEF were self-reported administered. The reason for the higher prevalence could be due to different in composition of study population and the methodology used.

However, the prevalence observed from current study is similar from that of the study done by Balde, NM et al [73] in Conakry (Guinea), whereby Erectile dysfunction concerned 90 patients

(48%), however the difference from the current study was on severity of ED, a severe form was observed in 54%, a moderate form in 35% and a mild form in 12%.

The the above quoted studies reveals a wide variation in prevalences not only between studies done in different continents but also between studies done within the same continent.The explanation for this large differences in the reported prevalence of ED among diabetic men could be due to differences in methodology and population characteristics.

Studies conducted in Europe/developed world are likely to report low prevalences as compared to those done in developing world due to the fact that in developed world DM may be early detected and blood sugar may be well controlled and therefore the chronic complications are prevented i.e differences in diabetic care or educated are more likely to deny ED.

Among methodological differences, various definitions were used until the National Institute of Health consensus conference in 1993 which established the definition of ED as 'inability to achieve or maintain an erection for satisfactory sexual intercourse' [1]. This definition was used in most subsequent studies until the development of the International Index of Erectile Function (IIEF)[15].

There are other explanations for the observed large differences in the reported prevalences: It has been shown that patients tend to underreport ED when questioned directly by their health-care providers[93]. Therefore, the uses of validated questionnaires such as IIEF that are either self-administered or administered by an objective third party interviewer are preferred. However, inadequate linguistic validation and cultural adaptation of the IIEF may have had an influence on the prevalences reported in studies conducted in different ethnic communities. The present study used the IIEF which was translated into Kiswahili; this could have solved the problem of inadequate linguistic and cultural adaptation because almost all of the participants understand Kiswahili very well.

Some of the prevalence studies which used IIEF did not exclude men who had no opportunity for sexual activity and categorized them as having ED, in this current study this effect was

taken care of. Some studies were conducted in tertiary care settings while others were population- or primary care-based studies with different severities and complication profiles which would have affected the prevalence rates. Lastly, the majority of prevalence studies have not distinguished between type 1 and type 2 diabetes; therefore, limited data are available to determine whether there are prevalence differences between the two major types of diabetes[77].

5.3 Comparison between prevalence of ED in diabetic male patients with that observed in the general population

There are no ED studies in Tanzanian general population, for comparison with corresponding prevalences among Tanzanian diabetic population. However the current study give ED prevalence similar to ED prevalences obtained from different studies of ED in the general population, worldwide. For instance the Massachusetts Male Aging Study (MMAS) reported a prevalence of 52% in men aged 40 to 70%[5].

Similarly, in a large cross-sectional study of 729 men aged 30 and above done in Singapore by Tan et al [29], the overall prevalence of erectile dysfunction was 51.3%. Of these, 23.2% had mild ED, 8.8% had moderate ED, and 19.3% had severe ED.

Contraly to the higher prevalence observed in the current study, Bai et al [28] determined the age-adjusted prevalence of ED in 3 big cities of China and found it to be 28.34% (mild 15.99%, moderate 7.14%, and severe 5.21%.

While it is not possible to do direct comparison due to different methodology and sampling technique, more other studies gave lower prevalent rate of erectile dysfunction than that reported in Singapore study and the current study with Thailand [30] (37.5% of men 40-70 years), Australia [31] (33.9% of men 40-69 years) and in Italy, Parazzini et al [32] interviewed 2,010 men aged 18 years or more from January 1996 to February 1997, found ED in 257(12.8%) individuals.

Moreover, similar to prevalent rate of ED observed in the current study, Berrada et al [33] conducted a population-based study in Casablanca, Morocco in 655 randomly selected men

with at least 25 years old, and found ED to be prevalent in 54%. In Nigeria and Egypt, the prevalence of ED in the general population is 57.4% and 63.6% respectively.

5.4 Prevalence and severity of other sexual function domains

This study showed that the prevalence of dysfunction for other sexual function domains were 62.5%, 54.2%, 49%, and 59.9% for sexual desire, orgasmic function, intercourse satisfaction, and overall satisfaction respectively. The prevalence of severe form was approximately 28% in each of these domains. Contrary, Malavige et al[54] who studied 253 men with type 2 diabetes randomly selected from clinic in Colombo, Sri Lanka; found, after excluding men with complete ED, that the prevalence of reduced libido was 64 (25%) and that of ejaculation disorder was 68 (40.2). The difference between prevalences observed in current study from this study could probably be due to difference in sample size, methodology and type of diabetic of patients studied.

5.5 Risk factors associated with erectile dysfunction

5.5.1 Age

In this study, the prevalence of ED increased statistically significant with age, with age groups 18 to 30 years (12.1 %,), 31 – 45 years (42.6%), 46 – 60 years (54.4%), and 61 years and above (78.0%) respectively ($p<0.001$). This finding is consistent with previous studies which were done among diabetic men [6, 53, 63, 68, 70, 72-74] which showed that ED is associated with age. Fedele et al[72] showed that, the prevalence of ED increased with age from 4.6% in men aged 20-29 to 45.5% in those aged ≥60 years (test for trend,$p=0.0001$).

Similar to the current study, Khatib et al[70] revealed that the prevalence of ED increased with age from 26.5% in men aged under 40years, to 90% in age group above 70years. Severity of ED increased with age as well.

Morever, similar to findings from this present study, Ngalyuka et al[74], Balde et al[73], and Mota et al[71] showed that the patients who presented with ED were significantly older.

5.5.2 Duration of diabetes

Similar to previous studies [6, 53, 63, 68, 70, 72-74], the current study showed that prevalence of ED was significantly higher in patient who presented with longer duration of diabetes of more than 10yrs (68.2%) than those who presented with duration of less than 10years (48%); p<0.001. Fedele et al[72] showed that patients with diabetes lasting 6-10 and 11-30years has a risk of 1.5 and 2.0 respectively as compared to those with diabetes for <5years.

5.5.3 Type of diabetes

In the present study, the prevalence of erectile dysfunction was significantly higher in T2DM (62.5%) than in T1DM (23%); p<0.001. Similarly, Fedele et al[72] showed that ED was more in T2DM than T1DM patients (OR=0.7, 95%CI= 0.6-0.8).

In contrast to this study, Mota etal [71] who studied 310 male diabetic patients observed that ED was not correlated to type of DM.

5.5.4 Glycemic control

Some cross-sectional studies have shown an association between poor glycemic control and ED[70, 72, 74, 94] while other studies did not find such an association[6, 71]; similarly, the current study did not show that association. In the current study, the prevalence of ED was high (56.0%) in patients with high levels of fasting blood glucose (> 7mmol/l) than those with normal levels (53.4%), and in patients with normal levels of glycosylated hemoglobin, HbA1c (≤7%); however, the observed differences was not statsticaly significant (p>0.05).

Fedele et al[72] showed a risk in poor control to be 2.3 while in good control to be 1.7. The reason to why poor glycemic control was not associated from the current study could probably due to the small sample size.

5.5.5 Hypertension, Hyperlipidemia, and obesity

Hyperlipidemia[72], hypertension[6, 54], and obesity[71] are conditions that often coexist with diabetes: these factors are also independent risk factors for ED among diabetic men.

Similar to previous studies [6, 54, 70, 74], in the present study, prevalence of ED in hypertensive patients was significantly higher (65.8%) than in nonhypertensive (44.2%); $p<0.001$. Khatib et al[70] and Ngalyuka et al[74] showed that patients with hypertension was associated with ED in male diabetics. Reasons for increased prevalence of ED in hypertensive group could be the drugs they utilize as well as other cormobid conditions they may be having e.g conditions like atherosclerosis.

Similar to other studies [71, 72], the prevalence of ED in present study was high among patients with high level of total cholesterol (>5.2mmo/l), and overweight (BMI = 25 – 29.9) although the observed difference was not statistically significant ($p>0.05$). Again, the prevalence of ED was high in patients with normal levels of HDL-c (0.9-2.00mmol/l), and LDL-c (≤3.34mmol/l), Triglcerides (≤2.3mmol/l but also the observed difference was not statisticaly significant ($p>0.05$). Mota et al[71] showed that obesity has a positive correlation with ED while dyslipidemia could not show correlation.

Lack of significance of these parameters in the present study could probably be due to small sample size.

5.5.6 Use of antihypertensive

Various medications, including several antihypertensive agents such as beta blockers[13] and thiazide diuretics[13] have been shown to have an additive deleterious effect on diabetic ED. Contraly to the present study, Balde et al[73] and Ngalyuka et al[74] showed that patients who presented with ED often took drugs for associated cardiovascular diseases. In the current study, the use of antihypertensive was not statistically significant associated with increased prevalence of ED with exception of Angiotensin Receptor Blockers (ARBs). Lack of association with other drugs as compared to previous study could probably be due to small number of individuals on those medications, in this study population majority (76.4%) of the patients were on ARBs. Significantly increased risk for ED on ARBs use could be due to the fact that these patients were on prior other anti-hypertensive because ARBs are not usually the first line antihypertensive in the population were this study was conducted. Other reason could be due to small sample size of studied population.

5.5.7 Other risk factors

The present study showed that other risk factors which was highly statistically significant associated with high prevalence of ED included history of cigarette smoking, history of drinking alcohol, past history of stroke, history of peripheral neuropathy and evidence of peripheral vascular disease ($p<0.05$). Similarly, previous studies showed that risk factors such as smoking[10, 68, 72] and excessive alcohol intake have been shown to have an additive deleterious effect on diabetic ED[55].

Similar to the present study, Fedele et al[72], Balde et al[73], Khatib et al[70], and Ngalyuka et al[74], found that patients with history of stroke, presence of peripheral neuropathy and evidence of peripheral arterial disease were associated with ED in male diabetics.

In 2003, Mota etal [71] , studied 310 male diabetic patients and observed that ED showed a positive correlation with stroke, arteriopathy and smoking, but it was not correlated alcohol consumption.

5.6 Multivariate logistic regression for predictors of ED

On Multivariate logistic regression the predictors of ED in the present study were found to be old age (odds ratio (OR) = 7.1, 95% CI 1.2-40.7), evidence of peripheral neuropathy (OR=5.9, 95% CI 1.6-21.3), and evidence of peripheral vascular disease (OR=2.5, 95% CI 1.2-5.3) {p= 0.028, 0.019, and 0.007 respectively}. Similarly, McCulloch et al[63] on using linear logistic models for analysis, of the five most significant association with ED, two were age ($p<0.001$), and symptomatic peripheral neuropathy ($p<0.001$).

In contrast, Fedele et al[72] showed, after taking into account the confounding role of age that ED was more in T2DM than T1DM, longer duration of DM, and poor glycemic control while Khatib et al[70] showed that apart from age other independent risk factors of ED were hypertension, glycemic control, coronary artery disease, retinopathy, and neuropathy. The difference between the present study and that of Fedele et al on this aspect could be due to differences in statistical analysis, in the current study the author could not adjust for age due to small sample size.

5.7 Comparison of risk factors associated with ED in general population with that in diabetic patients

The current study and other similar studies of ED done among diabetic male revealed similar risk factors associated with ED to that reported in the general population. The increased ED with age which was seen from the current study is similar to that in the general population. According to the Massachusetts Male Aging Study (MMAS), which surveyed 1790 men, the prevalence of ED increased with age [5].

Similar to the present study, in study done by Tan et al[29], the prevalence rate was similar amongst the 30-39 (47.1% had ED) and 40-49 (42.8%) age groups, the prevalence rate started to increase after the age of 50. The prevalence of ED increased significantly from 64.0 (50-59 years) through 77.4% (60-69 years) to 90.0% (70 years and above).

Similarly, in study done by Bai et al [28], the prevalence of ED increased with age. Cardiovascular disease, diabetes and hyperlipidaemia were positively correlated with the increased prevalence. Sociodemographic and lifestyle factors, such as education, spouse companionship, living conditions, cigarette and alcohol consumption or duration also have association with prevalence of ED.

Similar to the current study, Berrada et al [33] showed that ED increased with age, and was highly prevalent in the illiterate. Risk factors were diabetes, hypertension, heart disease, and smoking.

However, most of these association studies were cross-sectional and should be interpreted with caution.

5.8 Association between erectile dysfunction and other sexual function domains

In this study the prevalence of erectile dysfunction was statistically significant highly associated with other sexual domains. The study showed that the possibility of having normal sexual desire, normal orgasmic functions, intercourse satisfaction or overall sexual satisfaction

is almost zero (p<0001) i.e ED is closely related and a predictor of other sexual dysfunction domains.

In contrary, Balde et al [73] found that in 28% of the cases, erectile dysfunction was associated with a decline in libido and in 26% with ejaculation disorders while Malavige et al[54] found a strong association between ED and reduced libido (odds ratio [OR] = 4.38, 95 confidence interval[CI] = 1.39-13.82), and ejaculation disorders(OR = 4.41, CI = 2.08-9.39). The difference between prevalence observed in current study from that of Balde et al could be due to diffences in sample size, methodology, and type of DM of patients studied.

5.9 Use of medications for ED among diabetic male patients attending diabetic clinic at MNH

In this study, only 24(14%) of patients with ED ever tried medications for ED, of whom 22 (91.7%) used oral medications (Phosphodiasterase inhibitors) and 50% of them got satisfied with the medications.

5.10 This study revealed very low percentage of divorce despite high prevalence of erectile dysfunction, probable explanations for this observation could be: most of diabetic patients are educated on the possibilities of chronic complications of diabetes such as ED later in life and they therefore adapt to live as couple even if ED arise or in most cultural systems women are not allowed to leave their husband at all.

CHAPTER SIX

6 CONCLUSION AND RECOMMENDATIONS

6.1 CONCLUSION

This study showed that the prevalence of erectile dysfunction is high among DM patients. The study further showed that old age, peripheral neuropathy and evidence of peripheral arterial disease are main factors strongly associated with ED. It was also showed that ED is highly associated with dysfunction of other sexual domains. Very few patients ever tried use of medications for ED.

6.2 RECOMMENDATIONS

These findings are similar to those found in other studies done in different countries reinforcing the need to screen for ED in diabetic patients as it is done for other chronic complications of diabetes in routine clinical practices for early detection, treatment and possibly prevention. The screening for presence or absence of ED can be best done by the use of IIEF questionnaire. Interventions aimed at prevention of DM especially type 2; early diagnosis of DM and detection of its complications e.g ED, and adherence to treatment to prevent these complication should be implemented. ED is a neglected medical problem that warranty screening at each visit and deliver appropriate treatment. Further studies should emphasize on temporal variation to show true causality especially in low-income settings.

6.3 STUDY STRENGTH

In study, the interview on the IIEF questionnaire was done by the author among all participants; this increased the internal validity of the study.

Due to stigma attached to ED there was a potential that study participant could either shy away from participating or hide information related to ED. We minimized this by providing adequate information to the potential study participants on the importance of study and that we

will not judge them as person rather we want to understand ED for appropriate management for them and colleagues. We also ensured confidentiality.

6.4 STUDY LIMITATIONS

One of potential limitations is about generalizability. Muhimbili National Hospital might not be a true representative of the general population but probably relevant to the population of diabetics.

Other causes of ED such as depression were not explored in this study. Casuality can not be drawn from cross-sectional studies; because both exposure and outcome are assessed at the same point in time i.e this study cannot establish a temporal cause-effect relationship. However, it can confirm the association between cormobidity and ED in patients with diabetes.

Due to the consecutive nature of recruiting the study subjects may not be representative of true diabetic population (bias due to self selection).

7.0 REFERENCES

1. NIH Consensus Conference. Impotence.NIH Consensus Development Panel on Impotence. JAMA, 1993. 270(1): p. 83-90.

2. Neelima V. Chu and S.V. Edelman, Diabetes and Erectile Dysfunction. Clinical diabetes, 2001. 15: p. 63-71.

3. Kubin, M., G. Wagner, and A.R. Fugl-Meyer, Epidemiology of erectile dysfunction. Int J Impot Res, 2003. 15(1): p. 63-71.

4. Shiri, R., et al., Relationship between smoking and erectile dysfunction. Int J Impot Res, 2005. 17(2): p. 164-9.

5. Feldman HA, G.I., Hatzichriston DG, Krame RJ, McKinlazy JB Impotence and its medical and psychological correlates: results of the Massachusetts Male Ageing Study. J urol, 1994. 151(1): p. 54-61.

6. Siu SC, et al., Prevalence of and risk factors for erectile dysfunction in Hong Kong diabetic patients. Diabet Med 2001. 18: p. 732-738.

7. Deutsch S and S. L., Previously unrecognized diabetes mellitus in sexually impotent men. JAMA, 1980. 244: p. 2430-32.

8. Braun M, W.G., Klotz T, Reifennah B, Mathers M, Engelman U, Epidemiol of ED; results of the Cologne male survey. Int J of Impot Res 2000. 12: p. 305-11.

9. Marumo K, N.J., Murai M. , Age-related prevalence of erectile dysfunction in Japan: assessment by the International Index of Erectile Function. Int J Urol, 2001. 8 p. 53-59.

10. Nicolosi A, et al., Epidemiology of erectile dysfunction in four countries: Cross-national study of the prevalence and correlates of erectile dysfunction. Urology 2003. 61: p. 201–6.

11. ., M.E., Prevalence of erectile dysfunction: need for treatment? Int J Impot Res, 2002. 14 Suppl 1: p. S22-28.

12. Ryder, A.B.a.R.E.J., New Treatment Options for ErectileDysfunction in Patients with Diabetes Mellitus. Drugs, 2004. 64 (23): p. 2667-2688

13. Rosen, R., Sexual function assessment in male physiological and self-report measures Int J impotence Res, 1998. 10: p. 559-563.

14. Korenman, S., New insights into erectile dysfunction: a practical approach. Am J Med, 1998. 105: p. 125-144.

15. Rosen, R.C., J.C. Cappelleri, and N. Gendrano, 3rd, The International Index of Erectile Function (IIEF): a state-of-the-science review. Int J Impot Res, 2002. 14(4): p. 226-44.

16. Rosen, R.C., S.E. Althof, and F. Giuliano, Research instruments for the diagnosis and treatment of patients with erectile dysfunction. Urology, 2006. 68(3 Suppl): p. 6-16.

17. Broderick, G., Evidence based assessment of erectile dysfunction. Int J Impot Res, 1998. 10: p. 564-573.

18. Charles R. Moore, R.W., Pathophysiology and treatment of diabetic erectile dysfunction. Asian J Androl, 2006. 8(6): p. 675–68.

19. Thorve, V.S., et al., Diabetes-induced erectile dysfunction: epidemiology, pathophysiology and management. J Diabetes Complications.

20. Gray, H., Anatomy of the Human Body. 38th Edition. Philadelphia. 2000: p. 783-785.

21. de Groat, W.C. and A.M. Booth, Synaptic transimission in pelvic ganglia. In: Maggi C.A (Ed) ; The Autonomic Nervous system,vol 3, Nervous control of the urogenital system.1st Ed. Harward Academic publishers,London 1993: p. 291-347.

22. Kaiser FE, et al., Impotence and aging: clinical and hormonal factors. J Am Geriatr Soc, 1988. 36: p. 511-21.

23. Brock GB and L. TF, Drug-induced sexual dysfuction: an update. . Drug Safety, 1993. 8: p. 414-26.

24. McVary KT, C.S., Wessells H, Subcommittee on Smoking and Erectile dysfunction Socioeconomic Committee, Sexual Medicine Society of North America. Smoking and erectile dysfunction: evidence based analysis J urol 2001. 166(5): p. 1624-32.

25. Martin-Morales A, S.-C.J., Saenz de Tejada, Rodriguez-Vela L, Jiminez-Cruzz JF, Burgos-Rodriguez R, Prevalence and independent risk factors for erectile dysfunction in Spain: results from the epidemilogia de la disfuncion erectila masculina study. J Urol, 2001. 166: p. 569-74.

26. Ayta, I.A., J.B. McKinlay, and R.J. Krane, The likely worldwide increase in erectile dysfunction between 1995 and 2025 and some possible policy consequences. BJU Int, 1999. 84(1): p. 50-6.

27. McKinlay, J.B., The worldwide prevalence and epidemiology of erectile dysfunction. Int J Impot Res, 2000. 12 Suppl 4: p. S6-S11.

28. Bai, Q., et al., Prevalence and risk factors of erectile dysfunction in three cities of China: a community-based study. Asian J Androl, 2004. 6(4): p. 343-8.

29. Tan, J.K., et al., Erectile dysfunction in Singapore: prevalence and its associated factors--a population-based study. Singapore Med J, 2003. 44(1): p. 20-6.

30. Kongkanand, A., Prevalence of erectile dysfunction in Thailand. Thai Erectile Dysfunction Epidemiological Study Group. Int J Androl, 2000. 23 Suppl 2: p. 77-80.

31. Chew, K.K., et al., Male erectile dysfunction: its prevalence in Western australia and associated sociodemographic factors. J Sex Med, 2008. 5(1): p. 60-9.

32. Parazzini, F., et al., Frequency and determinants of erectile dysfunction in Italy. Eur Urol, 2000. 37(1): p. 43-9.

33. Berrada, S., et al., Prevalence of erectile dysfunction and its correlates: a population-based study in Morocco. Int J Impot Res, 2003. 15 Suppl 1: p. S3-7.

34. Afolayan, A.J. and M.T. Yakubu, Erectile dysfunction management options in Nigeria. J Sex Med, 2009. 6(4): p. 1090-102.

35. Gray, P. and B. Campbell, Erectile dysfunction and its correlates among the Ariaal of northern Kenya. Int J Impot Res, 2005. 17(5): p. 445-9.

36. Kapona YM, Y.C., Erectile dysfunction as seen in Dar-es-Salaam City. Tanzania Medical Journal, 2005. 20(2): p. 4-5.

37. Muller A, M.J., Cardiovascular disease, metabolic syndrome and erectile dysfunction. Curr Opin Urol, 2006 Nov. 16(6): p. 435-43.

38. Burnett, A.L., The role of nitric oxide in erectile dysfunction: implications for medical therapy. J Clin Hypertens (Greenwich), 2006. 8(12 Suppl 4): p. 53-62.

39. Guay, A.T., Relation of endothelial cell function to erectile dysfunction: implications for treatment. Am J Cardiol, 2005. 96(12B): p. 52M-56M.

40. Kloner, R., Erectile dysfunction and hypertension. Int J Impot Res, 2007. 19(3): p. 296-302.

41. Herbert, K., et al., The prevalence of erectile dysfunction in heart failure patients by race and ethnicity. Int J Impot Res, 2008. 20(5): p. 507-11.

42. Cerqueira, J., M. Moraes, and S. Glina, Erectile dysfunction: prevalence and associated variables in patients with chronic renal failure. Int J Impot Res, 2002. 14(2): p. 65-71.

43. Espinoza, R., et al., Prevalence of erectile dysfunction in kidney transplant recipients. Transplant Proc, 2006. 38(3): p. 916-7.

44. Wei, M., et al., Total cholesterol and high density lipoprotein cholesterol as important predictors of erectile dysfunction. Am J Epidemiol, 1994. 140(10): p. 930-7.

45. Ledda, A., Cigarette smoking, hypertension and erectile dysfunction. Curr Med Res Opin, 2000. 16 Suppl 1: p. s13-6.

46. Wong, S.Y., J.C. Leung, and J. Woo, A prospective study on the association between lower urinary tract symptoms (LUTS) and erectile dysfunction: results from a large study in elderly Chinese in Southern China. J Sex Med, 2009. 6(7): p. 2024-31.

47. Mbalamula, A., case control study of sexual dysfunction in patients with lower urinary tract symptoms at Muhimbili National Hospital. Master of Medicine dissertation. Muhimbili University College of Health and Allied Sciences, 2008.

48. Gunduz MI, Gumus BH, and S. C., Relationship between metabolic syndrome and erectile dysfunction. Asian J Androl, 2004. 6: p. 355–8.

49. Esposito K, G.F., Martedi E, Feola G, Marfella R, D'Armiento M, Giugliano D. Diabetes Care High proportions of erectile dysfunction in men with the metabolic syndrome. 2005. 28: p. 1201–3.

50. Heidler S, et al., Is the metabolic syndrome an independent risk factor for erectile dysfunction? J Urol, 2007. 177: p. 651–4.

51. Corona G, M.E., Schulman C, Petrone L,, C.A. Mansani R, Balercia G, Chiarini V, Forti, and M.M. G, Psychobiologic correlates of the metabolic syndrome and associated sexual dysfunction. Eur Urol 2006. 50: p. 595–604.

52. Ahn TY, P.J., Lee SW, Hong JH, Park NC,Kim JJ, Park K, Park H, Hyun JS. , Prevalence and risk factors for erectile dysfunction in Korean men:Results of an epidemiological study. J Sex Med, 2007. 4: p. 1269–76.

53. Cho NH, A.C., Park JY, Ahn TY, Lee HW,, K.I. Park TS, Pomerantz K, Park C, Kimm, and C.D. KC, Prevalence of erectile dysfunction in Korean men with Type 2 diabetes mellitus. Diabet Med, 2006. ;23: p. 198–203.

54. Malavige LS, J.S., Kathriarachchi ST, Sivayogan S, Fernando DJ, Levy JC., Erectile dysfunction among men with diabetes is strongly associated with premature ejaculation and reduced libido. . J Sex Med, 2008. 5: p. 2125–34.

55. Derby CA, et al., Modifiable risk factors and erectile dysfunction: Can lifestyle changes modify risk? Urology, 2000. 56: p. 302–6.

56. Byers, M., Erectile dysfunction: Hard facts and Treatment Options. British Columbia Persons with AIDS society, 2000.

57. Heyns CF, F.M., The urological management of Patients with AIDS. BJU Int, 2005. 95(5): p. 709-16.

58. Mkongo, E., The prevalence of erectile dysfunction and the associated risk factors among HIV patients in Dar-es-Salaam Care and Treatment Clinics. Master of Medicine dissertation. Muhimbili University College of Health and Allied Sciences, 2009.

59. Hatzichristou D, et al., Diagnostic steps in the evaluation of patients with erectile dysfunction. J Urol, 2002. 168: p. 615–20.

60. Spark RF, W.R., Connolly PB. , Impotence is not always psychogenic. Newer insights into hypothalamic-pituitary-gonadal dysfunction. JAMA 1980. ;243: p. 750–5.

61. N., M., Does testosterone have a role in erectile function? Am J Med 2006. 119: p. 373–82.

62. Sun, P., et al., Erectile dysfunction--an observable marker of diabetes mellitus? A large national epidemiological study. J Urol, 2006. 176(3): p. 1081-5; discussion 1085.

63. McCulloch, D.K., et al., The prevalence of diabetic impotence. Diabetologia, 1980. 18(4): p. 279-83.

64. Burchardt T, B.M., Karden J, et al., Reduction of endothelial and smooth muscle density in the corpora cavernosa of the streptozotocin induced diabetic rat. J Urol., 2000. 164(5): p. 1807-1811.

65. Shabsigh R, R.J., Aversa A, et al. , The evolving role of testosterone in the treatment of erectile dysfunction. Int J Clin Pract., 2006. 60(9): p. 1087-1092.

66. Shabsigh, R., Testosterone therapy in erectile dysfunction and hypogonadism. J Sex Med, 2005. 2(6): p. 785-92.

67. Stellato RK, F.H., Hamdy O, Horton ES, McKinlay JB. , Testosterone, sex hormone-binding globulin, and the development of type 2 diabetes in middle-aged men: prospective results from the Massachusetts male aging study. Diabetes Care., 2000. 23(4): p. 490-494.

68. Klein, R., et al., Prevalence of self-reported erectile dysfunction in people with long-term IDDM. Diabetes Care, 1996. 19(2): p. 135-41.

69. Lewis, R.W., Epidemiology of erectile dysfunction. Urol Clin North Am, 2001. 28(2): p. 209-16, vii.

70. Khatib, F.A., et al., Sexual dysfunction among Jordanian men with diabetes. Saudi Med J, 2006. 27(3): p. 351-6.

71. Mota M, L.C., Mota E, Panus C, Panus A. , Erectle dysfunction in DM. Rom Inter med, 2003. 41: p. 163-77.

72. Fedele, D., et al., Erectile dysfunction in diabetic subjects in Italy. Gruppo Italiano Studio Deficit Erettile nei Diabetici. Diabetes Care, 1998. 21(11): p. 1973-7.

73. Balde, N.M., et al., [Erectile dysfunction and diabetes in Conakry (Guinea): frequency and clinical characteristics from 187 diabetic patients]. Ann Endocrinol (Paris), 2006. 67(4): p. 338-42.

74. P.K.Ngalyuka, E.O.A., C.F.Otieno, J.K.Kayima. , The prevalence of erectile dysfunction and the associated risk factors in Kenyan men with type II diabetes at the Kenyatta national outpatient clinic. Master of Medicine dissertation. University of Nairobi, 2008. Available at www.uonbi.ac.ke/faculties /turntopdf.php? project_id=5288..October 5th, 2010.

75. Laumann, E.O., A. Paik, and R.C. Rosen, Sexual dysfunction in the United States: prevalence and predictors. JAMA, 1999. 281(6): p. 537-44.

76. Fonseca V, J.A., Endothelial and erectile dysfunction, diabetes mellitus, and the metabolic syndrome: Common pathways and treatments? Am J

Cardiol, 2005. 96: p. 13M–18M.

77. Malavige LS, a.L.J., Erectile dysfunction in diabetes mellitus. . J Sex Med 2009. 6: p. 1232–1247.

78. Urciuoli R, C.T., Carlinil M, Giuglietti M, Botti FM. , Prostaglandin E1 for treatment of erectile dysfunction. Cochrane Database Syst Rev, 2004. 2: p.:CD001784. DOI: 10.1002/14651858. CD001784.pub2.

79. Israilov S, S.J., Niv E, Engelstein D, Livne and B.J. P, Evaluation of a progressive treatment program for erectile dysfunction in patients with diabetes mellitus. Int J Impot Res, 2005. 17:: p. 431–6.

80. Nieschlag E, S.R., Behre HM, Gooren LJ, Kaufman JM, Legros JJ, Lunenfeld B, Morley JE, Schulman C,Wang C,Weidner W,Wu FC Investigation, treatment and monitoring of late-onset hypogonadism in males. ISA, ISSAM, and EAU recommendations. . .Eur Urol, 2005. 48: p. 1–4.

81. Leiblum SR, e., Principles and practice of sex therapy.4th edition .New York, London:. The Guilford Press, 2007. .

82. Strong TD, G.M., Burnett AL, Champion HC, Bivalacqua TJ. , Endothelium-specific gene and stem cell-based therapy for erectile dysfunction. Asian J Androl., 2008 Jan. 10(1): p.:14- 22.

83. Muammer, K., Patrick, E. T., Hunter, C. C., Wayne, J. G. H., & Trinity, J. B. , Gene therapy for erectile dysfunction: Fact or fiction? European Urology, 2006. 50: p. 1208−1222.

84. Atul, L., & Anoop, M. ;: and Erectile dysfunction in diabetic males: Plausible mechanisms and management strategies. Diabetes & Metabolic Syndrome. Clinical Research & Reviews, 2008. 2 p. 81−86.

85. Hartman, U., . The efficacy of psychosexual therapy for erectile dysfunction: A critical review of outcome studies. International Journal of Impotence Research, 1998. 10(3): p. S23.

86. Davidson, From experience gained in diabetic clinics, ED is a markedly under-diagnosed problem. Davidson's Principles and Practices of Medicine 20th edtn: p. 476.

87. United Republic of Tanzania. Bureau of Statistics. Population and Housing Report.

88. WHO., Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. WHO Technical Report, Geneva, 1995. Series 854.

89. WHO., International Society of Hypertension guidelines for the management of hypertension. J Hypertens, 1999. 17: p. 151-83.

90. Amstrong, D.G. and L.A. Lavery, Diabetic Foot Ulcers: Prevention, Diagnosis, and Classification. Am Fam Physician, 1998. 57(6): p. 1325-1332.

91. Moffatt C and O.H. L., Graduated compression hosiery for venous leg ulceration. J Wound Care, 1995. 4: p. 459-462.

92. Stacey MC, et al., The use of compression therapy in the treatment of venous leg ulcers: a recommended management pathway. . EWMA J 2002. 2(1): p. 9–13.

93. Litwin MS, et al., Differences in urologist and patient assessments of health related quality of life in men with prostate cancer: Results of the CaPSURE database. J Urol, 1998;. 159:: p. 1988–92.

94. Arie, R., Ofra, K., Yehuda, K., Ella, T., Juza, C., Tamar, S., & Itamar, R. , Prevalence and risk factors for erectile dysfunction in men with diabetes, hypertension, or both diseases: A Clinical Cardiologycommunity survey among 1,412 Israeli men. . Clinical Cardiology, 2003. 26: p. 25-30.

8.0 APPENDICES

8.1 APPENDIX I

8.1.1 QUESTIONNAIRE (ENGLISH VERSION)

Erectile dysfunction and risk factors among diabetic men at MNHl - 2011

Name of interviewer: _____ Date of interview: _____

Patients Initials: _____MNH File No: _____ DIC No: _____

SOCIODEMOGRAPHIC CHARACTERISTICS

Q **No.**	**Questions**	**Coding categories**	**Response**
Q 1	How old are you?	Age (years) _____	[ ]
Q 2	Marital status?	1. Married 2. Single 3. Divorced 4. Cohabiting 5. Widowed 6. Others_____	[ ]
Q 3	Highest level of education?	**1.** Informal/None **2.** Primary **3**. Secondary **4.** Post Secondary **5**. University **6.** Others (specify)	[ ]
Q 4	Current employment?	1. Farmer/peasant 2. Government employee 3. Private sector employee 4. Self employment 5. Others_____	[ ]

RISK FACTORS AND CLINICAL HISTORY

Q 5	Date of diagnosis	Date: _____	[ ]
Q 6	Type of DM(From records)	**1.** Type 1 **2.** Type 2	[ ]
Q 7	Current diabetes treatment	**1.** Metformin. **2.** Glibenclamide **3.** Chlorpropamide **4.** Pioglitazone **5**. Gliclazide **6**. Insulin inj **7.** Others (Specify)_____	[ ]
Q 8	Family history of DM?	1. Yes 2. No	[ ]
Q 9	Family history of HTN?	1. Yes 2. No	[ ]
Q 10	Are you known hypertensive?	1. Yes 2. No	[ ]
Q 11	If yes, on what current medications?	1. Beta blockers 2. ACE-inhibitors 3. ARB 4. Diuretics 5. Calcium channel blockers 6. Others	[ ]
Q 12	Have you ever smoked cigarettes?	1. Yes 2. No 3. Stopped	[ ]
Q 13	If yes, for how long?	_____years	[ ]
Q 14	If yes, how much	_____/day (quantify)	[ ]
Q 15	Do you take alcohol?	1. Current drinker 2. No	

		3. Stopped	[ ]
Q 16	If yes, for how long?	_____years	[ ]
Q 17	If yes, how much	_____/day (quantify)	
Q 18	History of? (from patient & records) **1= YES 2=NO 3=DON'T KNOW**	18.1 Stroke 18.2 Depression 18.3 LUTs 18.4 Past surgery (pelvic, urogenital….) 18.5 P/neuropathy (numbness, diabetic foot...)	18.1 : [ ] 18.2 : [ ] 18.3 : [ ] 18.4: [ ] 18.5: [ ]

MEASUREMENTS AND LABORATORY FINDINGS

Clinical Measurements

Q 19 Height _____mts Weight _____ kg BMI_____ kg/m^2

1=<18.5 2= 18.5-24.9 3=25.0-29.9 4= >30 [ ]

Q 20 blood pressures: SBP1_____ DBP1_____mmHg

SBP2_____ DBP2_____mmHg (after 5 min)

SBPAv_____DBP ave_____mmHg

1= ≥ 140/90 2= Normal [ ]

Biochemistry

Q 21) Blood glucose FBG _____mmol/l

Q 22) Serum triglycerides _____mmol/l

Q 23) HDL _____mmol/l

Q 24) LDL _____mmol/l

Q 25) Total cholesterol _____mmol/l

Q 26) Glycated Haemoglobin _____ %

Screening for p/ neuropathy and peripheral vascular disease ☐

Q 27) Monofilament testing score _____ 1=can feel 2= can't feel

Q 28) SBP of brachial artery _____mmHg

SBP of pedal artery _____mmHg ☐

Ankle Brachial Index Score _____ 1= <0.9 2= 0.9-1.3 3= >1.3

THE MODIFIED INTERNATIONAL INDEX OF ERECTILE FUNCTION (IIEF) QUESTIONNAIRE

1. Over the past 6 months, how do you rate your confidence that you can get and keep your erection? ☐

   (1) Very low (2) Low (3) Moderate (4) High (5) Very high

2. Over the past 6 months, how often were you able to get an erection during sexual activity? ☐

   (1) Almost never/never (2) Much <1/2 the time (3) About 1/2 the time

   (4) Much >1/2 the time (5) Almost always/always

3. Over the past 6 months, when you had erections with sexual stimulation, how often were your erections hard enough for penetration? ☐

   (1) Almost never/never (2) Much <1/2 the time (3) About 1/2 the time

   (4) Much >1/2 the time (5) Almost always/always

4. Over the past 6 months, when you attempted sexual intercourse, how often were you able to penetrate (enter) your partner? ☐

   (1) Almost never/never (2) Much <1/2 the time (3) About 1/2 the time

   (4) Much >1/2 the time (5) Almost always/always

5. Over the past 6 months, during sexual intercourse, how often were you able to maintain your erection after you had penetrated (entered) your partner? ☐

(1) Almost never/never (2) Much <1/2 the time (3) About 1/2 the time (4) Much >1/2 the time (5) Almost always/always

6. Over the past 6 months, during sexual intercourse, how difficult was it to maintain your erection to completion of intercourse? ☐

(1) Extremely difficult (2) Very difficult (3) Difficult (4) Slightly difficult (5) Not dif ☐

7. Over the past 6 months, how many times have you attempted sexual intercourse?

(1) 1to 2 attempts (2) 3-4 attempts (3) 5-6 attempts (4) 7-10 attempts (5) ≥11 attempts

8. Over the past 6 months, when you attempted sexual intercourse how often was it satisfactory for you? ☐

(1) Almost never/never (2) Much <1/2 the time (3) About 1/2 the time (4) Much >1/2 the time (5) Almost always/always

9. Over the past 6 months, how much have you enjoyed sexual intercourse? ☐

(1) Not enjoyable (2) Not very enjoyable (3) Fairly enjoyable (4) Highly enjoyable (5) Very highly enjoyable

10. Over the past 6 months, when you had sexual stimulation or intercourse how often did you ejaculate? ☐

(1) Almost never/never (2) Much <1/2 the time (3) About 1/2 the time (4) Much >1/2 the time (5) Almost always/always

11. Over the past 6 months, when you had sexual stimulation or intercourse how often did

you have the feeling of orgasm or climax (with or without ejaculation)? ☐

(1) Almost never/never (2) Much <1/2 the time (3) About 1/2 the time (4) Much >1/2 the time (5) Almost always/always

12. Over the past 6 months, how often have you felt sexual desire? ☐

(1) Almost never/never (2) Much <1/2 the time (3) About 1/2 the time (4) Much >1/2 the time (5) Almost always/always

13. Over the past 6 months, how would you rate your level of sexual desire? ☐

(1) Very low or none at all (2) Low (3) Moderate (4) High (5) Very high ☐

14. Over the past 6 months, how satisfied have you been with you overall sex life?

(1) Very dissatisfied (2) mod dissatisfied (3) About equally satisfied and dissatisfied (4) Moderately satisfied (5)Very satisfied

15. Over the past 6 months, how satisfied have you been with your sexual relationsh ☐ with your partner?

(1) Very dissatisfied (2) mod dissatisfied (3) About equally satisfied and dissatisfied (4) Moderately satisfied (5)Very satisfied

Scoring Algorithm for IIEF - All items are scored in 5 domains as follows:

Domain	Questions	Patient score	Scoring the domain	Clinical interpretation	Presence of ED: 1-YES, 2-NO
1. Erectile function	1, 2, 3, 4, 5, 6		25-30: no dysfunction 19-24: mild dysfunction 13-18:mod dysfunction 6-12: severe dysfunction		
2. Intercourse satisfaction	7, 8, 9		13-15: no dysfunction 10-12: mild dysfunction 7-9:mod dysfunction 3-6: severe dysfunction		
3. Orgasmic function	10, 11		9-10: no dysfunction 7-8: mild dysfunction 5-6:mod dysfunction 2-4: severe dysfunction		
4. Sexual desire	12, 13		9-10: no dysfunction 7-8: mild dysfunction 5-6: mod dysfunction 2-4: severe dysfunction		
5. Overall satisfaction	14,15		9-10: no dysfunction 7-8: mild dysfunction 5-6: mod dysfunction 2-4: severe dysfunction		

For those with erectile dysfunction

Q 29) Onset of ED:

(1) Within past 3 months (2) 3-12 months (3) More than 12 months

(4) Problematic since first sexually active

Q 30) Have you ever tried medication for treating ED? 1=Yes 2=No

Q 31) If yes, which medication among these?

(1) Oral medication e.g. Cialis, Viagra (2) Injectables

(3) Vacuum (4) Other……..

Q 32) were you satisfied with this medication (did it work)? 1= Yes 2 = No

8.1.2 QUESTIONNAIRE (KISWAHILI VERSION)

UPUNGUFU WA NGUVU ZA KIUME KWA WAGONJWA WA KISUKARI

Msimbo…….

Tarehe ya usaili……

Namba ya hospitali……..

TAARIFA BINAFSI

1. Umri…….
2. Hali ya ndoa
    1. Hujaowa
    2. Umeowa/unaishi na mpenzi

3. Umetaliki/umetengana

4. Mgane

3. Kiwango cha elimu

1. Hujasoma

2. Elimu ya msingi

3. Elimu ya sekondari

4. Elimu ya chuo

4. Kazi 1. Mkulima 2. Serikalini 3. Shirika bimafsi 4. Binafsi

SABABU ZA UPUNGUFU WA NGUVU ZA KIUME NA HISTORIA YA KIAFYA

5. Lini uligundulika kuwa mgonjwa wa kisukari…….

6. Aina ya kisukari

1= ya kwanza 2= ya pili

7. Dawa gani unatumia?

a) Glibenlamide

b) Chloropropamide

c) Metformin

d) Insulin

e) Nyinginezo…… (taja)

8. Kisukari katika familia

1= ndiyo 2= hapana

9. Shinikizo la damu katika familia 1=ndiyo 2=hapana

10. Una ugonjwa wa shinikizo la damu?

1= ndiyo 2= hapana

11. Kama ndiyo, dawa gani unatumia

a) beta blocker

b) ACE- inhibitors

c) Diuretics

d) Calcium channel blockers

e) Nyinginezo……..(taja)

12. Ulishawahi kuvuta sigara?

1= ndiyo 2= hapana 3= niliacha……(lini?)

13. Kama ndiyo, ulivuta kwa muda gani…….

14. Sigara ngapi kwa siku……

15. Unatumia kileo/pombe?

1= ndiyo 2= hapana 3= niliacha ……..

16. Kama ndiyo, ulitumia kwa muda gani……

17. Chupa/gilasi ngapi kwa siku…….

18.1 Ulishawahi kuugua ugonjwa wa kiharusi?

1= ndiyo 2= hapana

18.2 Ulishawahi kutibiwa kwa ugonjwa wa mfadahiko?

1= ndiyo 2= hapana

18.3 Matatizo ya kukojoa kwa taabu 1=ndiyo 2=hapana ……..

18.4 Ulishawahi kufanyiwa upasuaji…….(taja)

18.5 Kupata ganzi miguuni,kupata vidinda/ kukatwa mguu kwa sababu ya kisukari……

VIPIMO VYA MWILI

19. Urefu……..(cm)

Uzito……..(kg)

BMI…….(kg/m^2)

20. BP ya kwanza……(mmHg)

BP ya pili……(mmHg)

Wastani…… …(mmHg)

VIPIMO VYA MAABARA

21. Kiwango cha sukari…….mmol/l

22. Triglyceride……..mmol/l

23. HDL…….mmol/l

24. LDL.....mmol/l

25. Cholesterol ya jumla……mmol/l

26. Glycosylated Haemoglobin……%

KUPIMA UWEPO WA GANZI NA MATATIZO KATIKA MISHIPA YA DAMU

27. Monofilamenti 'score'……

28. Systolic BP ya ateri ya brachial……..

Systolic BP ya ateri ya pedal……..

Ankle Brachial Index Score…….

E. DODOSO SANIFU LA KIMATAIFA LA KUPIMA UWEZO WA KUDINDISHA (IIEF)

Katika kipindi cha miezi sita iliyopita:

1. Unatathminije uwezo wako wa muda wa kuanza kudindisha na kudumisha udindishaji huo? ☐
   (1) Uko chini sana (2) Uko chini (3) Wastani (4) Uko juu (5) Uko juu sana
2. Ni kwa kiasi gani umeweza kudindisha kila mara ulipokuwa unafanya mapenzi? ☐
   (1) Karibu haikuwezekana/Haikuwezekana kabisa
   (2) Mara chache chini ya nusu ya safari nilizofanya
   (3) Kama nusu ya safari nilizofanya
   (4) Mara nyingi zaidi ya nusu ya safari nilizofanya
   (5) Karibu kila mara/mara zote
3. Ulipodindisha kutokana na hamasa fulani, ni mara ngapi **udindishaji** wako ulikuwa imara kiasi cha kuweza **kupenyeza**?
   (1) Karibu haikuwezekana/Haikuwezekana kabisa

(2) Mara chache chini ya nusu ya safari nilizodindisha

(3) Kama nusu ya safari nilizodindisha

(4) Mara nyingi zaidi ya nusu ya safari nilizodindisha

(5) Karibu kila mara/mara zote

4. Ulipojaribu kufanya mapenzi, ni mara ngapi ulifanikiwa kupenyeza (kuingiza) kwa mwenzi wako?

(1) Karibu haikuwezekana/Haikuwezekana kabisa

(2) Mara chache chini ya nusu ya safari nilizojaribu

(3) Kama nusu ya safari nilizojaribu

(4) Mara nyingi zaidi ya nusu ya safari nilizojaribu

(5) Karibu kila mara/mara zote

5. Wakati kufanya mapenzi, ni mara ngapi uliweza kudumisha udindishaji wako baada ya kupenyeza (kuingiza) kwa mwenzi wako?

(1) Karibu haikuwezekana/Haikuwezekana kabisa

(2) Mara chache chini ya nusu ya safari nilizofanya

(3) Kama nusu ya safari nilizofanya

(4) Mara nyingi zaidi ya nusu ya safari nilizofanya

(5) Karibu kila mara/mara zote

6. Wakati wa kufanya mapenzi, ilikuwa vigumu kiasi gani kudumisha udindishaji wako hadi mwisho?

(1) Vigumu kupita kiasi (2) Vigumu sana (3) Vigumu (4) Vigumu kiasi (5) Hakuna ugumu

7. Ni mara ngapi umefanya mapenzi?

(1) Mara 1 hadi 2 (2) Mara 3-4 (3) Mara 5-6 (4) Mara 7-10 (5) Zaidi ya mara 11

8. Ulipofanya mapenzi, mara ngapi uliridhika?

(1) Karibu haikuwezekana/Haikuwezekana kabisa

(2) Mara chache chini ya nusu ya safari nilizofanya

(3) Kama nusu ya safari nilizofanya

(4) Mara nyingi zaidi ya nusu ya safari nilizofanya

(5) Karibu kila mara/mara zote

9. Kwa kiasi gani umefurahia kufanya mapenzi?

(1) Sikufurahia kabisa (2) Sikufurahia sana (3) Nilifurahia kiasi (4) Nilifurahia sana (5) Nilifurahia kupita kiasi

10. Ulipofanya tendo la ndoa au kupata hamasa, ni mara ngapi ulitoa shahawa?

(1) Karibu haikuwezekana/Haikuwezekana kabisa

(2) Mara chache chini ya nusu ya safari nilizofanya

(3) Kama nusu ya safari nilizofanya

(4) Mara nyingi zaidi ya nusu ya safari nilizofanya

(5) Karibu kila mara/mara zote

11. Ulipopata kupata hamasa ya kimapenzi au kufanya mapenzi, ni mara ngapi ulipata hisia za kufika kileleni (kwa kutoa au bila kutoa shahawa)?

(1) Karibu haikuwezekana/Haikuwezekana kabisa

(2) Mara chache chini ya nusu ya safari nilizofanya

(3) Kama nusu ya safari nilizofanya

(4) Mara nyingi zaidi ya nusu ya safari nilizofanya

(5) Karibu kila mara/mara zote

12. Mara ngapi umejisikia hamu ya kufanya mapenzi?

(1) Kama haijawahi kutokea/Haijawahi kutokea kabisa

(2) Mara chache chini ya nusu ya safari nilizojaribu kufanya

(3) Kama nusu ya safari nilizojaribu kufanya

(4) Mara nyingi zaidi ya nusu ya safari nilizojaribu kufanya

(5) Karibu kila mara/mara zote

13. Unatathminije kiwango chako cha hamu ya kufanya mapenzi?

(1) Kiko chini sana au hakuna kabisa (2) Kiko chini (3) Wastani (4) Kiko juu (5) Kiko juu sana

14. Unaionaje hali yako kwa ujumla kuhusiana na suala la kufanya mapenzi?

(1) Sijaridhika nayo kabisa (2) Sijaridhika kwa kiasi fulani (3) Niko nusu nusu (4) Nimeridhika kwa wastani (5) Nimeridhika sana

15. Unauonaje uhusiano wako wa kimapenzi na mwenzi wako?

(1) Sijaridhika nao kabisa (2) Sijaridhika kwa kiasi fulani (3) Niko nusu nusu (4) Nimeridhika kwa wastani (5) Nimeridhika sana

Uwiano wa Maswali ya IIEF Kimahesabu – Maswali yote yamewekwa katika maeneo makuu 5 kama ifuatavyo:

Eneo	Maswali	Alama ya Uwiano	Alama za eneo	Ufafanuzi wa Kitabibu	Uwepo wa Tatizo la Udindishaji (ED): 1-NDIYO, 2-HAPANA
1. Uwezo wa Kudindisha	1, 2, 3, 4, 5, 6		25-30: hakuna tatizo 19-24: tatizo dogo sana 13-18:tatizo la wastani 6-12: tatizo kubwa		
2. Uridhikaji katika Mapenzi	7, 8, 9		13-15: hakuna tatizo 10-12: tatizo dogo sana 7-9: tatizo la wastani 3-6: tatizo kubwa		
3. Uwezo wa Kufikia Kileleni	10, 11		9-10: hakuna tatizo 7-8: tatizo dogo sana 5-6: tatizo la wastani 2-4: tatizo kubwa		
4. Hamu ya Kufanya Mapenzi	12, 13		9-10: hakuna tatizo 7-8: tatizo dogo sana 5-6: tatizo la wastani 2-4: tatizo kubwa		
5. Hali ya Kuridhika kwa Ujumla	14,15		9-10: hakuna tatizo 7-8: tatizo dogo sana 5-6: tatizo la wastani 2-4: tatizo kubwa		

Kwa wenye upungufu wa nguvu za kiume tu

29. Upungufu wa nguvu za kiume ulianza lini

a) ndani ya miezi 3 iliyopita

b) miezi 3-12

c) zaidi ya miezi 12

d) tangu nianze kuwa na uwezo wa kufanya mapenzi

30. Ulishawahi kutumia dawa za kutibu ugonjwa wako?

1= ndiyo 2= hapana

31.Kama ndiyo, dawa gani?

a) vidonge k.m Cialis, Viagra

b) sindano

c) Vakyumu

d) Nyinginezo……..(taja)

32. Dawa zilikusaidia?

1= ndiyo 2=hapana

8.2 APPENDIX II

8.2.1 CONSENT FORM (ENGLISH VERSION)

MUHIMBILI UNIVERSITY OF HEALTH AND ALLIED SCIENCES DIRECTORATE OF RESEARCH AND PUBLICATIONS, MUHAS CONSENT FORM

CODE NO_____

CONSENT TO PERCIPATE IN THE STUDY ON ERECTILE DYSFUNCTION IN DIABETIC PATIENTS ATTENDING MNH DIABETIC CLINIC, IN 2011

Greetings,

My name is Dr Reuben Mutagaywa.

I am a postgraduate student in the Department of Internal Medicine

PURPOSE OF THE STUDY:

The study seeks to determine erectile dysfunction in Diabetic patients attending diabetic clinic at MNH

WHAT THE PARTICIPATION INVOLVES:

If you agree to participate in the study, you will be interviewed, and a detailed clinical history regarding your illness will be requested and a thorough physical examination will be done.

CONFIDENTIALITY:

The information you give in this research be treated great confidentially. All information collected on questionnaires will be entered into computer with an identification number. The information will be used only for the purpose of this research alone.

RIGHT TO WITHDRAW AND ALTERNATIVES:

Taking part in this study is completely your choice. If you choose not participate in the study, you will continue to receive all services that you would normally get from the clinic.

BENEFITS:

If you agree to take part in this study, and if you have erectile dysfunction, you will receive professional advice on how to deal with the situation.

RISKS:

There are no any risks in participating in this study.

Who to contact:

If you have any question about the study, you should contact Dr R. Mutagaywa, Tel no. 0717921555

If you have questions about your rights as participants, you may contact Prof A. Muhsin, Chairman of College research and Publication Committee. **P.O.Box 65001 Dar es Salaam, Tel 2150302-6**

Do you have any questions?

Do you agree?

Participant agrees…… Participant does NOT agree……..

I……have read the content of this form.

My questions have been answered. I agree to participate in this study

Signature participant……..

Signature witness……...

Date of signed consent……..

8.2.2 CONSENT FORM (KISWAHILI VERSION)

KUKUBALI KUSHIRIKI KWENYE UTAFITI

NAMBA YA SIRI_____

KUKUBALI KUSHIRIKI KATIKA UTAFITI WA UPUNGUFU WA NGUVU ZA KIUME KWA WAGONJWA WA KISUKARI, KLINIKI YA KISUKARI MUHIMBILI 2011

Salaam,

Mimi naitwa Dr Reuben mutagaywa.

Ni mwanafunzi wa uzamili katika idara ya magonjwa ya ndani, chuo kikuu cha tiba na sayansi za afya Muhimbili.

MADHUMUNI YA UTAFITI:

Kuchunguza tatizo la upungufu wa nguvu za kiume kwa wagonjwa wa Kisukari.

JINSI YA KUSHIRIKI:

Kama utakubali kushiriki, utafanyiwa usaili na kuulizwa maswali kuhusu historia ya ugonjwa wako na kufanyiwa uchunguzi wa mwili.

USIRI:

Taarifa zote utakazotoa kwenye utafiti huu zitatunzwa kwa usiri mkubwa. Taarifa zitakazokusanywa zitaingizwa kwenye kompyuta, zikiwa katika namba ya siri. Taarifa zitatumika kwa ajili ya utafiti huu tu.

HAKI YA KUJITOA KWENYE UTAFITI:

Kushiriki kwenye utafiti ni hiari yako.

Kama utachagua kutoshiriki, utaendelea kupata huduma za afya kutoka hospitalini kama hapo awali.

FAIDA:

Iwapo utakubali kushiriki katika utafiti huu na iwapo una tatizo la upungufu wa nguvu za kiume, utaweza kupata ushauri wa kitaalamu jinsi ya kukabiliana an tatizo hili.

ATHARI:

Hakuna athari zozote utakazozipata kutokana na ushiriki huu.

Nani wa kuwasiliana nae?

Endapo unahitaji kupata amelezo yoyote kuhusu utafiti, wasiliana na Dr R. Mutagaywa, simu namba 0717921555, na kuhusu haki za mshiriki wasiliana na Prof A. Muhsin, Mwenyekiti wa kamati ya utafiti P.O.Box 65001, Dar es Salaam, simu 2150302-6.

Una maswali?

Je unakubali kushiriki kwenye utafiti (weka alama)

Ndiyo……. Hapana……

Mimi……, nimeelezwa / nimesoma maelezo haya , maswali yangu yamejibiwa.

Nimekubali kushiriki kwenye utafiti huu

Sahii ya mgonjwa ……

Sahii ya ndugu/shahidi……

Sahii ya mtafiti……

Tarehe…….