PREDICTORS OF GLYCAEMIC CONTROL IN TYPE 2 DIABETES PATIENTS AT HELEN JOSEPH HOSPITAL DIABETIC CLINIC

Daniel Jacobus Roux

A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, in partial fulfillment of the requirements for the degree of Master of Medicine in the branch of Internal Medicine

Johannesburg, 2014

DECLARATION

I, Daniel Jacobus Roux declare that this research report is my own work. It is being submitted for the degree of Master of Medicine in the branch of Internal Medicine at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other University.

…… (Daniel Jacobus Roux) on this 20th day of February 2014

In loving memory of my father

Johan Roux

1943-2011

ABSTRACT

Background

Diabetes is a global epidemic. The International Diabetes Federation estimates that there are at least 285 million diabetics worldwide and this is estimated to grow to over 440 million by 2030 [1] . A study was conducted at the Helen Joseph Hospital Diabetic clinic in an attempt to identify predictors of glycaemic control and to compare the level of care to the 2012 Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) guidelines.

Methods

Patients were recruited from the Helen Joseph Hospital Diabetic clinic. To be included the patient had to be part of the coloured (mixed race) community, be willing to give informed consent, be older than 18 years, have an HBA_{1C} taken within 6 months, have a diagnosis of Type 2 diabetes mellitus and be a clinic attendee for at least 1 year. Pregnant patients, Type 1 diabetic patients, patients with a psychotic disorder or aphasia were excluded. Data collection consisted of face-to-face interviews, review of treatment, medication knowledge evaluation, a short examination and collection of recent blood results. Statistical analysis was done by stratifying patients into two groups by using the mean HBA_{1C}. Variables with a $p < 0.1$ from this analysis were used in a logistic regression model. In addition, the correlation between continuous variables were tested. A comparison was made between the level of care and the 2012 SEMDSA guidelines.

Results

A total of 100 patients were recruited into the study. The mean age was 62.8 years with mean duration of diabetes of 15.8 and clinic attendance of 10.9 years. The group had very poor education level and the median income of R1200 per month was also low. The mean HBA_{1C} was found to be 9.74%, well above the target recommended by SEMDSA. Knowledge of diabetes with respect to management and complications was very poor. Age > 50 years (OR 0.372 CI 0.06-2.26), estimated glomerular filtration rate ≥ 60 ml/min/1.73m^2 (OR 0.90 CI 0.25-3.27), experiencing a microvascular complication (OR 0.73 CI 0.11-5.07) or any other diabetic complication (OR 0.56 CI 0.07-4.38) and having experienced a hypoglycaemic episode (OR 0.31 CI 0.09-1.10) predicted better glycaemic control. Duration of diabetes < 10 years (OR 1.36 CI 0.37-5.02), diastolic blood pressure ≥ 70 mmHg (OR 2.80 CI 0.80-9.78), aspirin dosage ≥ 150 mg daily (OR 6.47 CI 1.60-26.05), simvastatin dosage = 40 mg daily (OR 2.35 CI 0.31-18.10) and body mass index > 25 kg/m^2 (OR 1.09 CI 0.49-2.41) all predicted a poorer glycaemic result.

HBA_{1C} was found to positively correlate with diastolic blood pressure ($p = 0.0024$, $r = 0.31$). Systolic blood pressure positively correlated with diastolic blood pressure ($p < 0.0001$, $r = 0.56$). Apart from correlating with systolic blood pressure and HBA_{1C}, diastolic blood pressure also positively correlated with the triglyceride level ($p = 0.0003$, $r = 0.36$). Positive correlations between total cholesterol, triglycerides, HDL-C and LDL-C were found. As expected, body mass index and waist circumference correlated positively ($p < 0.0001$, $r = 0.82$).

Level of care was not at the level recommended by the 2012 SEMDSA guidelines. Only 6% of patients met the waist circumference goal. Only 15% of patients achieved blood

pressure goal. Most of the patients (86%) who qualified for aspirin did not receive it. In the group of patients receiving aspirin 33% did not qualify. According to the SEMDSA guidelines, most of the patients not receiving a statin (90%) should have been on statin therapy. Only 23.5% of patients on statins were at lipid goal. The frequency of laboratory testing did not meet SEMDSA guidelines. There were 31 (31%) patients without a urea, creatinine and electrolyte test for the previous year and 37 (37%) patients without a lipogram for the previous year. Only 21 patients had a listed urine albumin/creatinine ratio and only 33% of these had been done in the previous year.

Conclusions

Various new variables were identified in the search for predictors of glycaemic control. It was surprising to find that education level, monthly income, smoking status and knowledge of diabetes did not have a statistical impact on glycaemic control. Increased age, duration of diabetes, glomerular filtration rate, hypoglycaemic frequency and diabetic complications experienced were associated with improved glycaemic control. Increased diastolic blood pressure, aspirin dosage, statin dosage and body mass index were associated with worse glycaemic control. The standard of care in the clinic was found on the whole to be inferior to the level of care recommended by SEMDSA.

ACKNOWLEDGEMENTS

Firstly, thank you Jesus Christ. You created me and gave me everything I needed to do this work.

A special thank you to Professor F.J.Raal, my supervisor. Without his help and guidance this project would not have been possible. I would like to thank him for always being available and for providing me with quick and positive feedback.

Prof. A. Bentley was of immense help. She was instrumental with respect to construction of the right questions to ask. I would like to thank her for her time, insight and enthusiasm.

Dr. Denise Evans and Arthemon Nguweneza did the statistical analysis. I cannot thank them enough. I thank them for all the p values, odd ratios and everything that goes with it. They have improved the "confidence interval" of the research significantly.

Lynne McNamara kindly proofread my thesis. Her assistance improved the quality of this work immensely. Her responses were quick and to the point. I am so incredibly fortunate to have crossed paths with her!

I would like to thank my lovely wife, Karin, and my baby girl, Kara. They supported me all the way. Time away from them is always hard.

Lastly I would like to thank the patients who participated. Without them this study would not have been possible.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

NOMENCLATURE

ACR	Albumin creatinine ratio
BMI	Body mass index
CI	Confidence Interval
CKD	Chronic kidney disease
cm	Centimeter
DASH	Dietary Approaches to Stop Hypertension
eg	For example
eGFR	Estimated Glomerular filtration rate
GORD	Gastro oesophageal reflux disease
GFR	Glomerular filtration rate
HbA_{1C}	Glycosylated haemoglobin
HIV	Human immunodeficiency virus
HDL	High density lipoprotein
HDL-C	High density lipoprotein cholesterol
HJH	Helen Joseph Hospital
IDF	International Diabetes Federation
IGT	Impaired glucose tolerance
IQR	Interquartile range
ie	id est
JNC7	Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure
kg	kilogram
LDL	Low density lipoprotein
LDL-C	Low density lipoprotein cholesterol
NHLS	National Health Laboratory System
OGTT	Oral glucose tolerance test
OR	Odds ratio
PCR	Protein Creatinine ratio
SD	Standard deviation
SEMDSA	Society for Endocrinology, Metabolism and Diabetes of South Africa
TC	Total cholesterol

TGL	Triglycerides
TIA	Transient ischaemic attack
U&E	Urea, creatinine and electrolytes
WHO	World Health Organisation

1. CHAPTER 1 – LITERATURE REVIEW

1.0. Introduction

Diabetes mellitus is a global epidemic. The International Diabetes Federation estimates that there are currently 285 million diabetics worldwide and this is estimated to grow to over 440 million by 2030 [1].

The global phenomenon of increased diabetes prevalence is also seen in South Africa. Helen Joseph Hospital is a tertiary hospital with 700 beds. During 2011 a total of 2808 patients were seen at the hospital's diabetes clinic, an mean of 234 patients per month. The diabetic clinic serves a specific cohort of diabetic patients being limited to patients with Type 1 and certain patients with Type 2 diabetes. The Type 2 diabetes patients are only accepted from other clinics if they are on insulin and not achieving adequate glycaemic control i.e. controlling blood sugar within certain targets. This population of patients seen at the diabetic clinic thus excludes the diabetic population attending the other clinics at the hospital and also those undiagnosed. In the United States it is estimated that one third of patients are not aware they are suffering from diabetes [2].

The economic cost of management of the disease is enormous and the long-term complications, both macro- and microvascular, are devastating. Macrovascular complications include stroke, ischaemic heart disease and peripheral vascular disease. Microvascular complications include diabetic nephropathy, diabetic retinopathy and diabetic neuropathy. Numerous studies have shown that improved glycaemic control can delay or even prevent the development of both micro- and macrovascular complications [3]. However, tight glycaemic control is often difficult to achieve.

Limited data has been published on predictors of glycaemic control. Currently there is no published South African data. The main aim of this study was to attempt to find factors predicting glycaemic control in the diabetic clinic population at Helen Joseph Hospital. A secondary aim was to correlate HBA_{1C}, blood pressure, lipids, body mass index and waist circumference and to assess the standard of care in the clinic with respect to the 2012 Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) guidelines for management of Type 2 diabetes.

In the following sections risk factors for predicting glycaemic control as found in the literature will be discussed. There will also be a brief focus on controlling blood pressure and dyslipidaemia as these are also important in securing the health of the diabetic patient. This will be followed by a critical review on current literature on guideline adherence with focus on the South African context.

1.1. Factors found in literature to predict glycaemic control

1.1.1. Factors worsening glycaemic control

Various factors in the literature have been cited as worsening control**.** Smoking, which increases insulin resistance and exacerbates the risk of cardiovascular disease in diabetics, long working hours and false perceptions of control and treatment in the Spanish speaking population of Northeast Colorado have all been cited [18, 64, 65].

Caffeine ingestion worsens control, especially when taken postprandially. This is in contrast to the protective effect of caffeine for developing diabetes, but only a few

small studies have been done [43] . The ingestion of soda drinks may also worsen control [66] .

Depressive symptoms worsen HbA_{1c} values in Type 1 diabetics but not Type 2 diabetics. However adherence to diet and exercise falls in both groups when depressive symptoms are present [67] .

1.1.2. Factors improving glycaemic control

Various factors have been researched and found to improve glucose control. Weight loss has been suggested [11] . Some authors argue that as little as 5% weight loss can improve glucose control [68] . Weight loss ironically, is also a beacon of poor control as it is one of the symptoms of diabetes [69] .

In addition, physical activity also improves glucose control. Most studies recommend at least 150 minutes of moderate exercise per week [68] . It seems that patient support groups can play a positive role in enforcing physical activity [70] . A higher level of education is a strong predictor of increased activity if the patient has available time [71] . The intensity of the exercise does not seem to play a role when the same amount of calories are burned [72] .

Lifestyle modification and continuous lifestyle intervention programmes have been shown to improve control [14,73 , 74] . Lifestyle modification seems to be effective in the management of diabetes irrespective of family history [75] .

Diabetes education has been positively linked with good control. In a study among Turkish immigrant diabetic patients poor compliance indicators included duration of diabetes and poorer knowledge of diabetes [76].

Age, motivation and an increase in diabetes knowledge level were all cited in one article as improving control [77]. Pharmacological therapy was also shown to be of benefit [68].

Proper nutrition, consisting of a low-fat reduced calorie diet has been suggested as improving glucose control [68, 78].

Self-monitoring of blood glucose in Type 2 diabetes, home monitoring by a public health nurse and follow-up phone call interventions have all been shown to be of benefit [79 - 80].

A moderate amount of alcohol ingestion, visual display of HbA_{1c} values with target values, providing primary care physicians with an electronic system guiding decision-making with respect to diabetic control and learned resourcefulness all played positive roles in glucose control [66 81 - 82].

Physician attitude at the time of diagnosis strongly influences the patient's perception of the seriousness of the disease, and this can have a positive effect on their control [83].

1.2. Factors influencing blood pressure and lipid control

As far as control in diabetic patients is concerned, glucose control is not the only target. Hypertension, dyslipidaemia and microalbuminuria must also be targeted [78].

Hypertension increases the risk of both macro- and microvascular complications in diabetes [84]. The latest Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) guidelines published in 2012, support a target blood pressure between 140/80 and 120/70 mmHg based on the latest data available [84]. In patients with primary or essential hypertension pharmacological intervention and lifestyle modification play a role. The 7th report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure (JNC7), which will be updated by the JNC8 later this year, lists the following lifestyle factors as being beneficial in the management of hypertension: weight loss in overweight patients, following the Dietary Approaches to Stop Hypertension (DASH) plan, a reduction in dietary sodium, regular aerobic physical activity and limitation of alcohol intake [85].

Dyslipidaemia also plays a major role in the macrovascular complications of diabetes and is a major risk factor for the development of atherosclerosis [86]. There is strong evidence supporting the notion that reducing total cholesterol (TC) and low density lipoprotein cholesterol (LDL-C) reduces the risk of cardiovascular disease [87]. Type 2 diabetes is considered a coronary artery disease risk equivalent by some authors implying that dyslipidaemia should be treated aggressively in this cohort of patients [86].

Apart from pharmacological intervention in managing dyslipidaemia, lifestyle intervention should always be encouraged. The strongest driving dietary factor is dietary

saturated fatty acids. Dietary trans-fat, also called unsaturated fat, increases LDL-C [87]. In contrast, increased dietary fibre intake reduces serum cholesterol [87]. Weight loss is beneficial but the effect is small, with only a 0.2 mmol/l drop in LDL-C for every 10kg of weight lost [87]. The effect of exercise on LDL-C is even smaller and less than the effect achieved by weight loss [87].

Triglyceride levels are lowered by a reduction of excessive body weight, reduction in alcohol use and in the intake of carbohydrates (mono- and disaccharides) and an increase in physical activity and ingestion of n-3 polyunsaturated fat which can be found in fish [87].

High density lipoprotein (HDL) is thought to be protective against cardiovascular disease. A reduction in dietary trans-fat, an increase in physical activity, a reduction of excessive body weight, a reduction in dietary carbohydrates, and when utilizing carbohydrates, choosing ones with a low glycaemic index and high fibre content, all increase HDL levels. Alcohol should also be used in moderation and smoking should be discouraged [87].

1.3. Diabetes guideline adherence with focus on the South African experience

The Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) publishes guidelines every three to 4 years on the management of type 2 diabetes mellitus. The most recent guidelines were published in 2012.

Guidelines are the best practice available in managing patients based on the latest published literature. Guideline adherence improves outcome. In 2004, Distiller published data confirming that guideline adherence is possible and showed that patients did benefit

[88] . He showed an impressive 90% reduction in hospital admissions of diabetic patients after implementation of strict guideline adherence [88] .

Adherence to guidelines worldwide is poor. The Centre of Disease control found that less than 5% of diabetes patients in the United States received care comparable to that prescribed by the American Diabetes Association guidelines [89] .

Some work has been done with respect to guideline adherence in South Africa. Just recently, while this study was ongoing, Okoroma, Harbor and Ross published a paper concluding that there was poor compliance with guidelines at a Kwazulu-Natal hospital [90] . Their comparison was made to the 2009 SEMDSA guidelines. Eighty three percent of patients had poor HbA_{1C}'s, lipid tests were rarely performed and foot exams were done in only 6% of the patients.

2. CHAPTER 2 – MATERIALS AND METHODS

2.0. Study objective

Limited literature is available on predictors of glycaemic control in the diabetic population and as far as I am aware there is no published South African data on this topic. The main aim of this study was an attempt to find predictors of glycaemic control in a cohort of diabetic patients attending the Helen Joseph Hospital diabetic clinic.

The main objectives of the study were:

1. To describe the diabetic population at Helen Joseph Hospital diabetic clinic included in the sample with respect to demographics, glucose, blood pressure and lipid control as well as knowledge and perception of diabetes.
2. To compare diabetic patients below the mean HbA_{1C} found in this study to those above the mean in order to find factors that may predict glycaemic control.
3. To correlate HbA_{1C} with blood pressure, lipids, body mass index and waist circumference.
4. To compare the level of care at the clinic to the recommendations of the 2012 SEMDSA guidelines.

2.1. Methods

2.1.1. Data source

This was an observational, cross sectional study. One hundred patients were recruited from the Helen Joseph Hospital diabetic clinic. The sample size was calculated using the statistical package Epi Info using a 2-sided confidence interval of 95%, a power of 80%, a ratio of unexposed to exposed (factors predicting good

control) of 4:1, percentage of unexposed with outcome 10% and percentage of exposed with outcome 50%. With these parameters the software calculated a required sample size of 67. As sample size can merely be predicted using statistical mathematics, the decision was taken to increase the sample size to 100 patients in an attempt to ensure statistical significance at the end of the study.

To be included in the study the patients had to satisfy the following criteria: be part of the coloured (mixed race) community, be able and willing to give informed consent, be older than 18 years, have an HbA_{1C} taken within 6 months of the interview date, have a diagnosis of Type 2 diabetes for at least 1 year and have been followed up at Helen Joseph Hospital diabetes clinic for at least 1 year. This study focused on this population group in an attempt to reduce cultural bias. The coloured community is the largest population group attending the clinic. This would potentially facilitate patient recruitment.

Patients who were pregnant, known to have Type 1 diabetes, have a known psychotic disorder or who had suffered a stroke and subsequent aphasia were excluded from the study.

Ethics approval was obtained from the Human Research Ethics Committee (Medical) of the University of the Witwatersrand, Clearance Certificate No M120844 (Appendix A).

2.1.2. Data collection

Data was collected at the Helen Joseph Hospital diabetic clinic. The clinic is open to patients every Wednesday, except for public holidays. On selected Wednesdays the clinic is open to Type 1 diabetic patients only. These clinic days were not used to recruit Type 2 diabetic patients.

All patients included in the study had to give informed consent to partake in the study. The informed consent document is listed in Appendix B. Informed consent was obtained after the patient reviewed the patient information leaflet. This document can be viewed in Appendix C.

Patients partaking in the study were seen early in the morning, long before the start of the diabetic clinic. After the data was collected for the research study I also managed their regular clinic consultation. They thus benefitted by leaving the clinic earlier than on a regular clinic day.

Data collection consisted of face-to-face interviews, review of the patients file for their latest treatment, an evaluation of their medication knowledge and collection of their latest blood results. They were also questioned and examined for evidence of end organ damage ie peripheral vascular disease, stroke, ischaemic heart disease, diabetic retinopathy, diabetic neuropathy and diabetic retinopathy.

All the data was collected and recorded by myself using the database software package Filemaker Pro© during the interviews. A patient data collection sheet can

be viewed in Appendix D. The following information was recorded during the face-to-face interviews:

2.1.2.1. Patient demographics

The following was recorded under patient demographics:

1. Date and time of interview.
2. Patient study number.
3. Age.
4. Gender.
5. Education level (No School, Primary, Secondary, Tertiary).
6. Highest education level (specific grade or degree).
7. Helen Joseph Hospital diabetic clinic attendance duration in years.
8. Duration of diabetes in years.
9. Co-morbidities. A co-morbidity score was also calculated comprising the sum of the patient's co-morbidities. A co-morbidity was listed if the patient volunteered it on questioning or if it was found recorded in the patient's file.

2.1.2.2. Patients opinion about the risk of diabetes

The patient was asked how serious they viewed diabetes as a disease. Possible responses were: Life threatening, very serious, serious, not so serious, not serious at all or not answered.

2.1.2.3. Smoking

The following smoking history was recorded:

1. Their smoking status: non-smoker, previous smoker, smoker, secondary smoker ie a patient exposed to passive smoke from another individual or previous secondary smoker.
2. The amount of cigarettes smoked per day for smokers and secondary smokers.
3. The amount of years smoked for smokers and secondary smokers.
4. The duration stopped in years was recorded if they had stopped smoking.

2.1.2.4. Questions on employment, income and living conditions

1. Patients were asked if they were employed or not. If they were employed they were also asked if they did night shifts as this is listed in the literature as a predictor of poor glycaemic control. [64]
2. They were asked what their net income was per month and also asked how many people were supported with that income. The average income per person was calculated by taking the net income per month and dividing that by the number of individuals supported by that income.
3. Their type of accommodation was recorded.
4. The number of bedrooms and people living in their dwelling were recorded to ascertain the population density.
5. They were asked if they had a refrigerator, hot water and toilet facilities.

6. Lastly the patient was asked if they had Internet access in an attempt to evaluate learned resourcefulness.

2.1.2.5. Questions on diabetes management, control and knowledge

1. Patients were asked how they would rate their overall health. They had to pick one from the following scale: Excellent, very good, good, average or poor.
2. Patients were asked to mention things they can do every day to improve their glycaemic control. This was an open question and they were given time to elaborate. An "improve glucose score" was calculated by adding the things they were able to mention.
3. Patients were asked to name things that are checked at the clinic to ensure that they are healthy diabetics. This was an open question and they were given no leads. If they mentioned any parameter ie cholesterol, blood pressure, glucose on clinic day, HBA_{1C}, weight or renal function they were asked to comment on the level of that parameter ie if it was high, normal or low for them. They could also say that they were not sure and could also choose not to answer the question. A "clinic check score" was calculated by adding all the things they could mention together.

The following values were used to divide the parameters into high, normal and low categories:

Table 1: Defining parameter categories

Parameter	Low	Normal	High
Total cholesterol (mmol/l)	< 4.5	< 4.5	≥ 4.5
Blood pressure (mmHg)*	SBP< 120* DBP < 70**	120 ≥ SBP < 140 > 70 DBP ≤ 80	SBP ≥ 140 DBP ≥ 80
Glucose (mmol/l)	< 4	4 – 8	> 8
HBA_{1C} (%)	< 6.5	6.5 – 7.5	> 7.5
Weight assessed according to BMI (kg/m^2)	< 22	22-25	> 25
Renal function assessed with eGFR ($ml/kg/1.73m^2$)	< 90	≥ 90	≥ 90

*SBP: Systolic blood pressure, **DBP: Diastolic blood pressure, ^eGFR: Estimated Glomerular filtration rate

4. Patients were asked if they thought they weighed too much for their height. Possible responses were yes, no, do not know and not answered.
5. Patients were asked how often they exercised. This was quantified as exercise for the purpose of doing exercise. A walk to the mall to do their regular grocery shopping or house cleaning did not qualify. The type of exercise was not recorded. Possible responses were daily, 6,5,4,3, twice or once a week, less often than above, do not exercise at all and not answered.
6. Patients were asked to name all possible complications of diabetes. This was an open question. They could mention any of the macrovascular complications ie stroke, transient ischaemic attack, ischaemic heart disease and peripheral vascular disease; microvascular complications ie diabetic retinopathy, nephropathy

and neuropathy or diabetic coma complications (coma because of hypoglycaemia or hyperglycaemia). A macrovascular complication score, microvascular complication score, diabetic coma score and total complication score were calculated by adding all the individual complications mentioned.

7. Asking them if they received diabetes education and if they visited a dietician in the previous year concluded this section.

2.1.2.6. Questions on hypo- and hyperglycaemia

Patients were asked to mention any symptoms of hypoglycaemia (anxiety, dizziness, coma, confusion, nausea, palpitations, shakiness, sweating, weakness, seizures, pallor) and hyperglycaemia (coma, dizziness, fatigue, polyuria, polydipisia, polyphagia). These included ones they might have experienced and symptoms they knew about. The questions were open and the patients were given time to respond. Subsequently a "hypoglycaemic knowledge score" as well as a "hyperglycaemic knowledge score" were calculated by adding all the correct answers that were given together.

The patients were asked how many times they experienced symptoms of hypoglycaemia in the previous year. The answer was categorized into the following: Daily, more than once a week, weekly, once every 2 weeks, once every 3 weeks, monthly, every other month, every 3 months, every 3-6months, less than 6 months, once a year, less than once a year, never and not answered.

2.1.2.7. Questions on glucose monitoring

The patient was asked if they used a glucometer. If they did have one they were asked to disclose how often they checked their glucose. Glucose checking frequency options included: Daily, 2-3 times per week, weekly, monthly, check it when I do not feel right or not answered.

In the next phase of the data acquisition, the patient was questioned about their current treatment.

2.1.2.8. Medication identification ability

An attempt was made to assess patient compliance. A selection of medication was placed on a table in the examination room and the patients asked to select their medication from this. There was no insulin on the table but they were also questioned on their insulin type and dosage. The insulin thus also formed part of the final calculation.

They got a mark for every tablet they could identify correctly. They lost marks for every tablet that was wrongly identified ie picked from the table but not on their prescription. The medication identified correctly was calculated as follows:

$$\frac{\text{(Medication correctly identified} - \text{medication incorrectly identified)}}{\text{Total amount of medication taken by the patient presented on the table}}$$

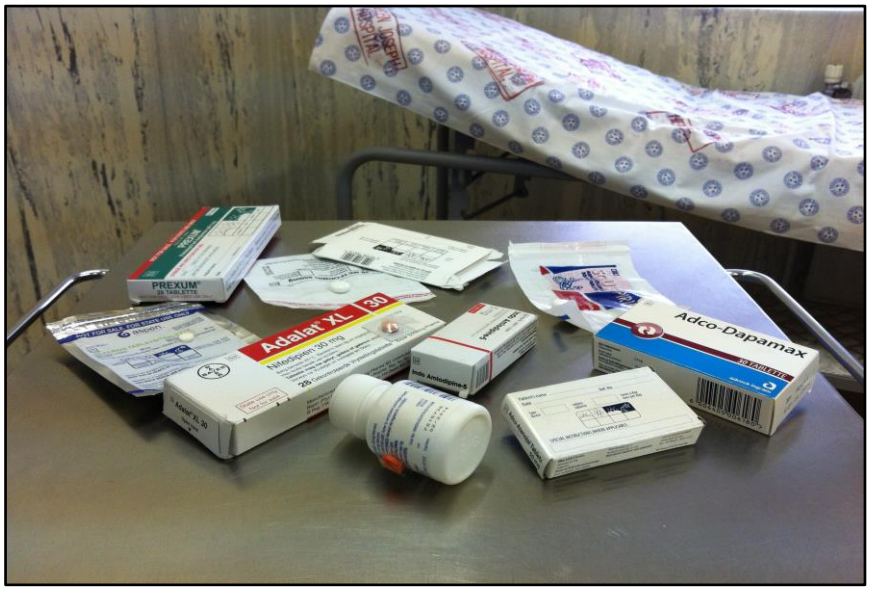

Figure 1: Medication used in clinic to assess compliance

The drugs that were used to assess medication identification ability were the following: amlodipine 5 mg, adalat XL (nifedipine) 30 mg, aspirin 300 mg, atenolol 50 mg, atenolol 100 mg, enalapril 10 mg, gliclazide 80 mg, indapamide 2.5 mg, metformin 850 mg and perindopril 4 mg.

2.1.2.9. Barriers to taking treatment

Patients were asked to mention any barriers to taking their treatment. Things like side effects, work schedule, forgetfulness etc. could be mentioned here.

2.1.2.10. Treatment

The patient's current treatment was recorded in the database.

The next phase of data acquisition entailed a brief examination which included the following:

2.1.2.11. Height

Height was measured using a stadiometer supplied by the Modern Scale Co (Pty) Ltd, (Johannesburg, South Africa). Patients were asked to stand upright after removing their shoes and all hair attire before the measurement.

2.1.2.12. Weight

Weight was measured using a standard calibrated scale. The scale was placed on a flat firm surface. Patients were requested to remove their shoes and any heavy clothing. The patients were not allowed to hold onto any object whilst on the scale.

2.1.2.13. Waist circumference

Waist circumference was measured using a tape measure. Patients were requested to remove all heavy clothing. Measurement was done with the patient standing and taken in a horizontal plane, midway between the inferior margin of the ribs and the superior border of the iliac crest. This method is recommended by the International Diabetes Federation (IDF) and can be accessed on their website (http://www.idf.org).

2.1.2.14. Neck circumference

Neck circumference was measured using a measuring tape. All obstructing clothing was removed before the measurement. Measurement was done with the patient standing.

2.1.2.15. Body mass index

Body mass index was calculated using the following formula:

$$\frac{\text{Weight (kg)}}{\text{Height (m)}^2}$$

2.1.2.16. Blood pressure

Blood pressure was taken using a calibrated automatic blood pressure monitor. Blood pressure was taken with the patient seated and feet on the floor, back supported, arm bared and resting on a surface at heart level. Before measurement patients were seated quietly for at least 5 minutes. An appropriately sized cuff (encircling 80% of the arm) was used. This is in accordance with the guidelines stipulated in the JNC7 and the South African hypertension guidelines [85, 91].

Although none of the patients were specifically asked to stop smoking, not to consume caffeine containing beverages or eat in the previous 30 minutes as recommended by the South African hypertension guidelines, it is believed that this was not the case for any of the patients in this study [91].

Mean blood pressure was calculated with the following formula:

(Systolic blood pressure + 2*Diastolic blood pressure)/3

2.1.2.17. Examination for diabetes complications

If the patient mentioned any macro- or microvascular complication suffered or if it was found during examination it was recorded.

Macrovascular complications included any history or clinical examination suggestive of stroke, transient ischaemic attack (TIA), ischaemic heart disease or peripheral vascular disease. Peripheral vascular disease was recorded as a complication if there was evidence of claudication, previous amputation or absent foot pulses.

Microvascular complications included any history or clinical examination suggestive of diabetic retinopathy (with direct ophthalmoscopy in the clinic or diagnosed by an ophthalmologist), diabetic nephropathy (eGFR < 60 ml/kg/1.73m^2 or evidence of microalbuminuria), diabetic neuropathy (if the patient was complaining of a burning sensation in their feet or could not detect vibration from a 128Hz tuning fork on the medial part of the first tarsal bone of either foot), autonomic neuropathy (if the patient volunteered any symptom which was consistent, eg impotence).

A "complication score" was calculated for macro- and microvascular complications suffered. A "total complication score" was also calculated to determine the total amount of complications the patient had experienced. Each complication suffered or experienced would add a point to the patient's complication score.

After the brief physical examination, the patients laboratory results were recorded.

2.1.2.18. Laboratory results

The following laboratory results were recorded with their respective dates.

1. Glucose measured by finger prick on the day of the clinic visit.

2. HBA_{1C} – If the HBA_{1C} value listed on the laboratory system was older than 6 months, an HBA_{1C} was taken on the day of the clinic visit to satisfy the inclusion criteria of the study.
3. Lipid profile.
4. Sodium, potassium, urea and creatinine.
5. Any urine protein examination listed on the laboratory system ie urine albumin/creatinine ratio, urine protein/creatinine ratio. Preference was given to albumin/creatinine ratios as these are recommended in the 2012 SEMDSA guidelines [92] .

2.2. Statistical analysis

Patient demographics and clinical characteristics were described and summarized. Patients were stratified into two groups: HbA_{1C} above 9.74 % and HbA_{1C} less than or equal to 9.74 % (above and below the mean HbA_{1C} for the sample n=100). Groups were compared using Student t test (for normally distributed or parametric data) or Kruskal–Wallis (for not normally distributed or non-parametric data) for continuous variables and Chi-square ($\chi 2$) test for proportions, where appropriate. A $p < 0.05$ was considered significant. In addition, the correlation between continuous variables was tested and the correlation coefficients and p values are presented.

Logistic regression models were used to test the association between the different variables and failure to control HbA_{1C}, defined as an HbA_{1C} above 9.74 % vs an HbA_{1C} less than or equal to 9.74 %. Odds ratio (OR) and 95% confidence interval (CI) are also presented. Variables identified in the bivariate analysis at $p < 0.1$ were considered in the regression model. In addition alternative definitions of failure to control HbA_{1C} including

an HbA_{1C} above 8 % and an HbA_{1C} above 7 % in sensitivity analyses were tested [3]

. Correlations were done between HBA_{1C}, blood pressure (systolic, diastolic), lipids (total and LDL-C, trigycerides as well as HDL-C), body mass index and waist circumference. Analyses were performed using the SAS® 9.1 statistical software package (SAS Institute, Inc., North Carolina, USA) and STATA™ 10.1 (StataCorp, Texas, USA).

3. CHAPTER 3 – RESULTS

The data collection for the study started on 3 October 2012 and was concluded on 27 February 2013, a total span of 168 days. One hundred patients were recruited. The data was independently analysed and verified by Dr. Denise Evans and Arthemon Nguweneza.

3.0. Description of the study population

3.0.1. Demographics

Table 2: Demographics of the study population

Characteristic		Sub category	All patients (n=100)	Sub category numbers
Age (years)	Mean (±SD)		62.8 (±9.7)	
Gender	n (%)	Male		28 (28%)
	n (%)	Female		72 (72%)
Duration of diabetes (years)	Mean (±SD)		15.8 (±9.4)	
Diabetic clinic attendance (years)	Mean (±SD)		10.9 (±7.4)	
Education level	n (%)	Primary		12 (12%)
	n (%)	Secondary		83 (83%)
	n (%)	Tertiary		5 (5%)
High school education level (Secondary education group)	n (%)	Std 6		18 (18%)
	n (%)	Std 7		9 (9%)
	n (%)	Std 8		28 (28%)
	n (%)	Std 9		11 (11%)
	n (%)	Std 10		15 (15%)
Total number of co-morbidities	Mean (±SD)		1.8 (±1.1)	
Co morbidity numbers	n (%)	0		6 (6%)
	n (%)	1		36 (36%)
	n (%)	2		33 (33%)
	n (%)	3		17 (17%)
	n (%)	4		6 (6%)
	n (%)	5		1 (1%)
	n (%)	6		1 (1%)

3.0.2. Smoking

Table 3: Smoking in the study population

Characteristic		Sub category	All patients (n=100)	Sub category numbers
Smoking status	n (%)	Non smoker		31 (31%)
	n (%)	Smoker		26 (26%)
	n (%)	Secondary smoker		9 (9%)
	n (%)	Previous smoker		35 (35%)
	n (%)	Previous secondary smoker		1 (1%)
Smoker: Pack years smoked (years)	Mean (±SD)		21.5 (±15.9) (n=26)	
Previous smoker: Duration stopped (years)	Mean (±SD)		18.1 (±14.9) (n=35)	

3.0.3. Employment, income and living conditions

Table 4: Employment, income and living conditions

Characteristic		Sub category	All patients (n=100 unless otherwise stated)	Sub category numbers
Employment	n (%)	Employed		14 (14%)
	n (%)	Not employed		86 (86%)
Monthly income known (R)	Mean (±SD)		3247 (±3984) (n=96)	
Mean income per person known (R)*	Mean (±SD)		1566 (±1724) (n=96)	
Type of accommodation	n (%)	Flat		22 (22%)
	n (%)	House		77 (77%)
	n (%)	Old age home		1 (1%)
Number of bedrooms in living unit	Mean (±SD)		2.5 (±0.8)	
Bedroom numbers	n (%)	1		11 (11%)
	n (%)	2		34 (34%)
	n (%)	3		45 (45%)
	n (%)	4		10 (10%)
Number of people per bedroom	Mean (±SD)		1.5 (±0.9)	
People per room numbers	n (%)	<1		15 (15%)
	n (%)	>=1-2		58 (58%)
	n (%)	>=2-3		19 (19%)
	n (%)	>=3-4		7 (7%)
	n (%)	>=4		1 (1%)
Hot water	n (%)		Yes	91 (91%)
	n (%)		No	9 (9%)
Internet access	n (%)		Yes	15 (15%)
	n (%)		No	85 (85%)

* Mean income per person = Monthly income / number of dependants

All the patients had refrigeration and toilet facilities.

3.0.4. Measurements

Table 5: Measurements of study population

Parameter		All patients (n=100)
Body mass index (kg/m^2)	Mean (±SD)	32.5 (±6.4)
Waist circumference (cm)	Mean (±SD)	106.4 (±15.0)
Neck circumference (cm)	Mean (±SD)	37.8 (±3.7)
Systolic blood pressure (mmHg)	Mean (±SD)	144.5 (±24.2)
Diastolic blood pressure (mmHg)	Mean (±SD)	78.0 (±13.0)
Mean blood pressure (mmHg)	Mean (±SD)	100.2 (±14.7)

The waist circumference and BMI for the male and female groups were as follows:

Table 6: BMI and waist circumference - males and females

Parameter		Male (n=28)	Female (n=72)
Body mass index (kg/m^2)	Mean (±SD)	30.13 (± 7.40)	33.37 (± 5.70)
Waist circumference (cm)	Mean (±SD)	106.89 (±20.45)	106.13 (±12.37)

When omitting the male individual with the largest waist circumference (186cm) and BMI (54.90 kg/m^2), the mean waist circumference and BMI of the male group dropped to 103.96 ± 13.6 cm and 29.21 ± 5.69 kg/m^2 respectively. The males on average thus had smaller BMIs and waist circumferences compared to the females.

3.0.5. Laboratory results

Table 7: Laboratory results of study population

Parameter		All patients (n=100)	Laboratory reference range (NHLS)
Glucose on clinic day (mmol/l)	Mean (±SD)	10.4 (±4.8)	3 – 7
HBA_{1C} (%)	Mean (±SD)	9.74 (±2.2)	< 6
Total cholesterol (mmol/l)	Mean (±SD)	4.6 (±1.3)	
Triglycerides (mmol/l)	Mean (±SD)	2.1 (±1.4)	0.5 – 1.5
HDL (mmol/l)	Mean (±SD)	1.2 (±0.3)	
LDL (mmol/l)	Mean (±SD)	2.4 (±1.0)	
Urea (mmol/l)	Mean (±SD)	6.9 (±3.8)	2.6 – 7.0
Estimated glomerular filtration rate (ml/min/1.73m^2)	Mean (±SD)	83.1 (±33.1)	> 90

Table 8: Urine albumin creatinine ratio (ACR) of patients in study population (21 available)

Parameter		
Urine ACR (mg/mmol) All patients (n=21)	Mean (±SD)	3.8 (±4.8)
Urine ACR (mg/mmol) Males (n=6)	Mean (±SD)	5.56 (±7.22)
Urine ACR (mg/mmol) Females (n=15)	Mean (±SD)	3.03 (±3.49)

Table 9: Urine PCR of patients in the study population (47 available)

Parameter		n=47
Urine PCR (g/mmol)	Mean (±SD)	0.08 (±0.22)

3.1. Comparing the group with HBA_{1C} (%) below mean to the group above the mean in order to find predictors of glycaemic control

Part of the inclusion criteria for the study was the availability of a recent HBA_{1C}, ie one taken within 6 months of the research clinic date. The mean time between the clinic research date and the HBA_{1C} date was 81.8 (±64.15) days (Mean (±SD). The maximum time was 181 days or 5 months and 28 days.

In the following sections tables summarizing the results after grouping the patients into 2 groups using the mean HBA_{1C} (9.74%) as divider will be shown. Variables with $p < 0.1$ were deemed significant enough to include in a logistic regression model.

3.1.1. Demographics

Table 10: Demographic data grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Age (years)	Median (IQR)	63 (58-70)	64 (60-72)	60 (52-69)	0.045*
Gender					0.171**
Male	n (%)	28 (28%)	19 (33%)	9 (21%)	
Female	n (%)	72 (72%)	38 (67%)	34 (79%)	

*T-Test, ** Chi-square test

Table 11: Demographic data grouped by HBA_{1C} - continued

Parameter		All patients	HBA_{1C} below mean (<= 9.74%)	HBA_{1C} above mean (>9.74%)	p value
		(n=100)	n=57	n=43	
Education level					0.733**
Primary	n (%)	12 (12%)	7 (12%)	5 (12%)	
Secondary	n (%)	83 (83%)	48 (84%)	35 (81%)	
Std 6	n (%)	18 (18%)	10 (18%)	8 (19%)	0.585**
Std 7	n (%)	9 (9%)	4 (7%)	5 (12%)	
Std 8	n (%)	28 (28%)	16 (28%)	12 (28%)	
Std 9	n (%)	11 (11%)	5 (9%)	6 (14%)	
Std 10	n (%)	15 (15%)	12 (21%)	3 (7%)	
Tertiary	n (%)	5 (5%)	2 (4%)	3 (7%)	
Duration of diabetes (years)	Median (IQR)	15 (9-21)	17 (10 – 24)	13 (8 – 18)	0.012^
Diabetes clinic attendance (years)	Median (IQR)	10 (5-15)	10 (5 – 18)	10 (5 –13)	0.105^
Co-morbidities					
Hypertension	n (%)	86 (86%)	49 (86%)	37 (86%)	0.991**
Dyslipidemia	n (%)	33 (33%)	18 (32%)	15 (35%)	0.728**
Ischaemic heart disease	n (%)	10 (10%)	4 (7%)	6 (14%)	0.252**
Osteoarthritis	n (%)	8 (8%)	6 (11%)	2 (5%)	0.284**
Hypothyroidism	n (%)	6 (6%)	3 (5%)	3 (7%)	0.721**
GORD	n (%)	5 (5%)	2 (4%)	3 (7%)	0.431**
Total number of co-morbidities					
0	n (%)	6 (6%)	3 (5%)	3 (7%)	0.629**
1	n (%)	36 (36%)	21 (37%)	15 (35%)	
2	n (%)	33 (33%)	16 (28%)	17 (40%)	
3	n (%)	17 (17%)	10 (18%)	7 (16%)	
4	n (%)	6 (6%)	5 (9%)	1 (2%)	
5	n (%)	1 (1%)	1 (2%)	0 (0%)	
6	n (%)	1 (1%)	1 (2%)	0 (0%)	

*T-Test, ^Kruskal Wallis test, ** Chi-square test

Age (p=0.045) and duration of diabetes (p=0.012) both had a $p < 0.1$.

Increasing age is cited in the literature as a factor that improves control [77].

Both gender (p=0.171) and duration of diabetes clinic attendance (p=0.105) was found not to be significant in this study.

A higher level of education was cited in the literature to relate to increased physical activity and hence better glycaemic control [71] . In this study, education level, even after sub-analysis of high school education, was not found to be a significant predictor of glycaemic control (p=0.733).

Low-grade systemic inflammation has been cited as a risk factor for developing diabetes [1] . Low-grade systemic inflammation would only have a bearing on inflammatory co-morbidities like rheumatoid arthritis. In this study, co-morbidities were not found to be predictors of glycaemic control. This was still the case after coding for the most prevalent co-morbidities, volunteered by the patient or recorded in the file ie hypertension (n=86), dyslipidaemia (n=33), ischaemic heart disease (n=10), osteoarthritis (n=7), hypothyroidism (n=6) and gastro oesophageal reflux disease (n=5). The total number of co-morbidities per patient also did not code as a predictor of glycaemic control.

The following co-morbidities, presented in decreasing frequency, were volunteered by the patient or recorded in the patient's file: hypertension (n=86), dyslipidemia (n=33), ischaemic heart disease (n=10), osteoarthritis (n=7), hypothyroidism (n=6), gastro-oesophageal reflux disease (n=5), asthma (n=4), gout (n=4), stroke (n=4), congestive cardiac failure (n=3), atrial fibrillation (n=2), chronic kidney disease (n= 2), epilepsy (n=2), multinodular goitre (n= 2), previous alcohol abuse (n=2), psoriasis (n=2), benign prostate hypertrophy (n=1), chronic obstructive airways disease (n=1), colon cancer in remission (n=1), deep venous thrombosis on lifelong warfarin (n=1), diverticulosis (n=1), general anxiety disorder (n=1), hay fever (n=1), pancreatitis (previous 2nd to alcohol) (n=1), peripheral vascular disease

(n=1), primary hyperparathyroidism (n=1), rheumatoid arthritis (n=1), stasis eczema 2nd to varicose veins (n=1) and subclinical hypothyroidism (n=1).

In summary the demographics of this study group shows a picture of an elderly group (mean ± standard deviation of 62.8 ± 9.7 years) with a long history of diabetes (15.8 ± 9.4) years. The average education level of the group was very low. Most of them had secondary education (83%) but when this was examined further only 15% of the group had matriculated. Only 5% of the study population had any after school training. The patients in the group had a mean co-morbidity count of 1.8 co-morbidities per patient.

3.1.2. Opinion about diabetes risk

Table 12: Opinion about diabetes grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Opinion about diabetes risk					0.930**
Not serious	n (%)	19 (19%)	11 (19%)	8 (19%)	
Life threatening or serious	n (%)	81 (81%)	46 (81%)	35 (81%)	

** Chi-square test

The patient's personal opinion about the risk of diabetes was found not found to be significant (p=0.930).

3.1.3. Smoking

Table 13: Smoking grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Smoking status					0.837**
Non smoker	n (%)	31 (31%)	19 (33%)	12 (28%)	
Smoker or Secondary smoker	n (%)	34 (34%)	19 (33%)	15 (35%)	
Previous smoker or Previous secondary smoker	n (%)	35 (35%)	19 (33%)	16(37%)	
Pack years smoked: smokers (years)	Median (IQR)	18 (10-29)	14 (4 – 29)	10 (8 – 20)	0.471^
Duration since stopped smoking: previous smokers and secondary smokers (years)	Median (IQR)	20 (5-28)	12 (6 – 30)	10 (5 – 25)	0.285^

^Kruskal Wallis test, ** Chi-square test

Smoking increases insulin resistance according to a study done in 2009 [18] . This was not supported by the data in this study. Smoking status, duration smoked and duration stopped were not statistically significant in predicting glycaemic control. There was also no evidence to suggest that glycaemic control was influenced by being a secondary or previous secondary smoker.

3.1.4. Employment, income and living conditions

Table 14: Employment, income and living conditions grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Employment					0.991**
Employed	n (%)	14 (14%)	8 (14%)	6 (14%)	
Not employed	n (%)	86 (86%)	49 (86%)	37 (86%)	
Mean income per person known (R)	Median (IQR)	1200 (600-1750)	1200 (600-1750)	1666 (700-1700)	0.677^
Type of accommodation					0.219**
Flat	n (%)	22 (22%)	10 (18%)	12 (28%)	
House	n (%)	77 (77%)	47 (82%)	30 (70%)	
Old age home	n (%)	1 (1%)	0 (0%)	1 (2%)	
Number of bedrooms					0.986**
1	n (%)	11 (11%)	6 (11%)	5 (12%)	
2	n (%)	34 (34%)	20 (35%)	14 (33%)	
3	n (%)	45 (45%)	25 (44%)	20 (47%)	
4	n (%)	10 (10%)	6 (11%)	4 (9%)	
Number of people per bedroom					0.186**
<1	n (%)	15 (15%)	6 (11%)	9 (21%)	
>=1-2	n (%)	58 (58%)	33 (58%)	25 (58%)	
>=2-3	n (%)	19 (19%)	11 (19%)	8 (19%)	
>=3-4	n (%)	7 (7%)	6 (11%)	1 (2%)	
>=4	n (%)	1 (1%)	1 (2%)	0 (0%)	
Hot water					0.187**
Yes	n (%)	91 (91%)	50 (88%)	41 (95%)	
No	n (%)	9 (9%)	7 (12%)	2 (5%)	
Does the patient have access to internet services?					0.381**
Yes	n (%)	15 (15%)	7 (12%)	8 (19%)	
No	n (%)	85 (85%)	50 (88%)	35 (81%)	

^Kruskal Wallis test, ** Chi-square test

Being employed (p=0.991) and the mean income per person (p=0.677) were found not to be statistically significant. It must be noted that the median income per person was only R1200.00 per month with an interquartile range of R600.00-R1750.00. This low income would significantly limit the participant's ability to follow a proper diabetic diet and implement lifestyle modifications necessary. These values also suggest that most of the patients had similar income.

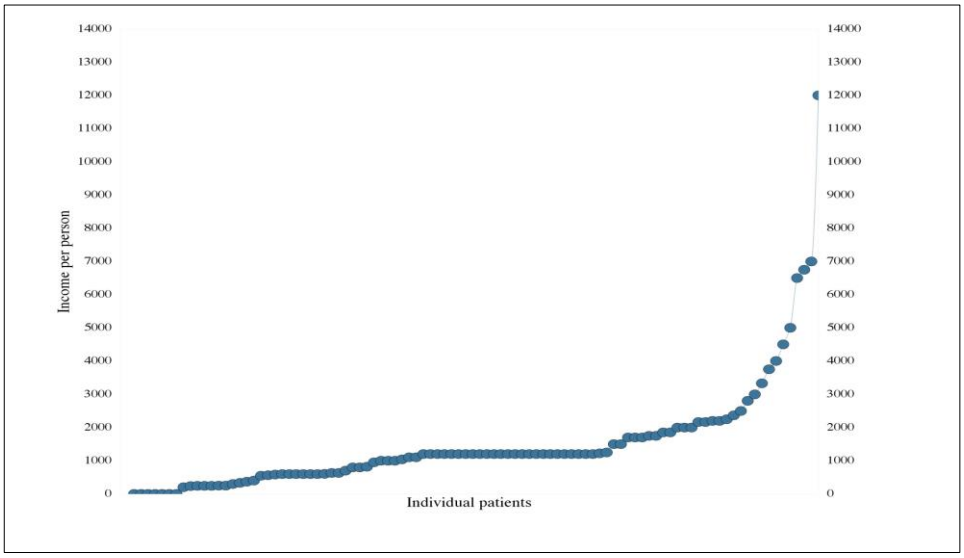

Figure 2: Mean income per person

Neither type of accommodation (p = 0.219), number of people per bedroom (p = 0.186) or having hot water or not (p = 0.187) were statistically significant enough to be included in the model.

Learned resourcefulness, which was assessed in this study by questioning the patient about Internet access, was also not found not to be statistically significant (p=0.381) [82] .

3.1.5. Diabetes management, control and knowledge

Table 15: Rating of own health grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
How would you rate your health?					0.806**
Excellent	n (%)	1 (1%)	0 (0%)	1 (2%)	
Very well	n (%)	7 (7%)	4 (7%)	3 (7%)	
Good	n (%)	35 (35%)	21 (37%)	14 (33%)	
Average	n (%)	49 (49%)	28 (49%)	21 (49%)	
Poor	n (%)	8 (8%)	4 (7%)	4 (9%)	

** Chi-square test

The patients rating of their own health (p=0.806) was not statistically significant enough to include in the model.

Table 16: Diabetes knowledge of ways to improve glucose grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Knowledge on ways to improve glucose at home					
Diet	n (%)	82 (82%)	48 (84%)	34 (79%)	0.507**
Exercise	n (%)	62 (62%)	35 (61%)	27 (62%)	0.887**
Glucose monitoring	n (%)	5 (5%)	3 (5%)	2 (5%)	0.889**
Medication	n (%)	33 (33%)	18 (32%)	15 (35%)	0.728**
Interventions (i.e. weight loss or stopping smoking)	n (%)	13 (13%)	5 (5%)	8 (8%)	0.148**
Patient number for each improved glucose score total (adding items together)					0.496**
0	n (%)	6 (6%)	4 (7%)	2 (5%)	
1	n (%)	22 (22%)	14 (25%)	8 (19%)	
2	n (%)	45 (45%)	24 (42%)	21 (49%)	
3	n (%)	25 (25%)	13 (23%)	12 (28%)	
4	n (%)	2 (2%)	2 (4%)	0 (0%)	

** Chi-square test

The patients knowledge of ways to improve glucose at home were not statistically significant enough to include in the model (p=0.496).

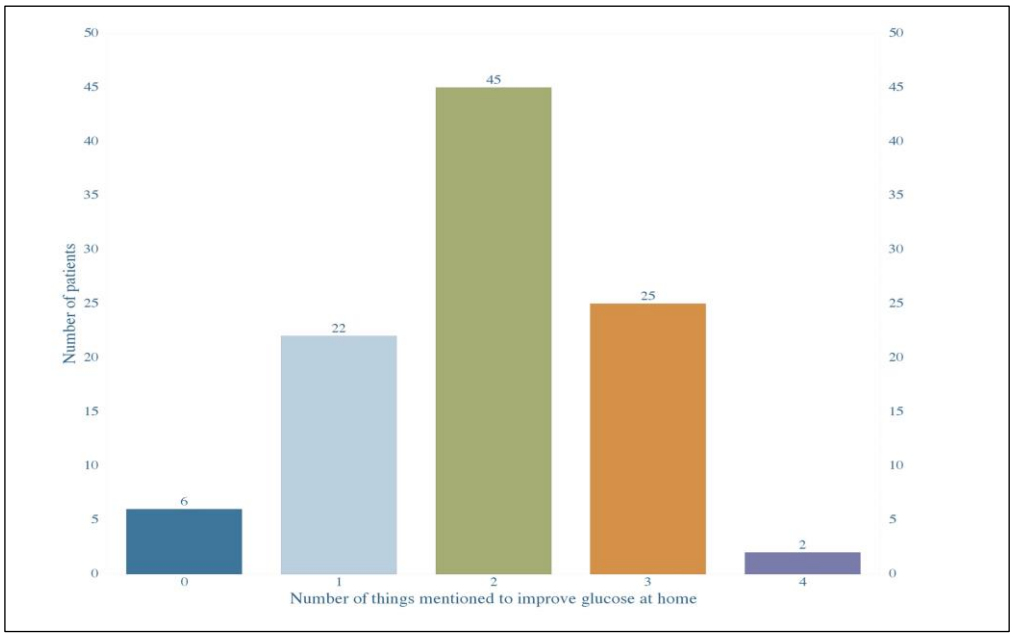

Figure 3: Patient numbers versus ways known to improve glucose at home

In the above figure it can be seen that 6% of the patients could not mention anything which might improve their glucose control. Less than half of the group (45%) could mention 2 things to improve glucose control at home.

Table 17: Diabetes knowledge of clinic checks known grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Things to check in clinic to ensure they are healthy diabetics					0.975**
0	n (%)	22 (22%)	14 (25%)	8 (19%)	
1	n (%)	15 (15%)	9 (16%)	6 (14%)	
2	n (%)	23 (23%)	13 (23%)	10 (23%)	
3	n (%)	27 (27%)	14 (25%)	13 (30%)	
4	n (%)	9 (9%)	5 (9%)	4 (9%)	
5	n (%)	4 (4%)	2 (4%)	2 (5%)	

** Chi-square test

Knowledge of things checked in the clinic to make sure that they are healthy diabetics were not found to be significant (p=0.975).

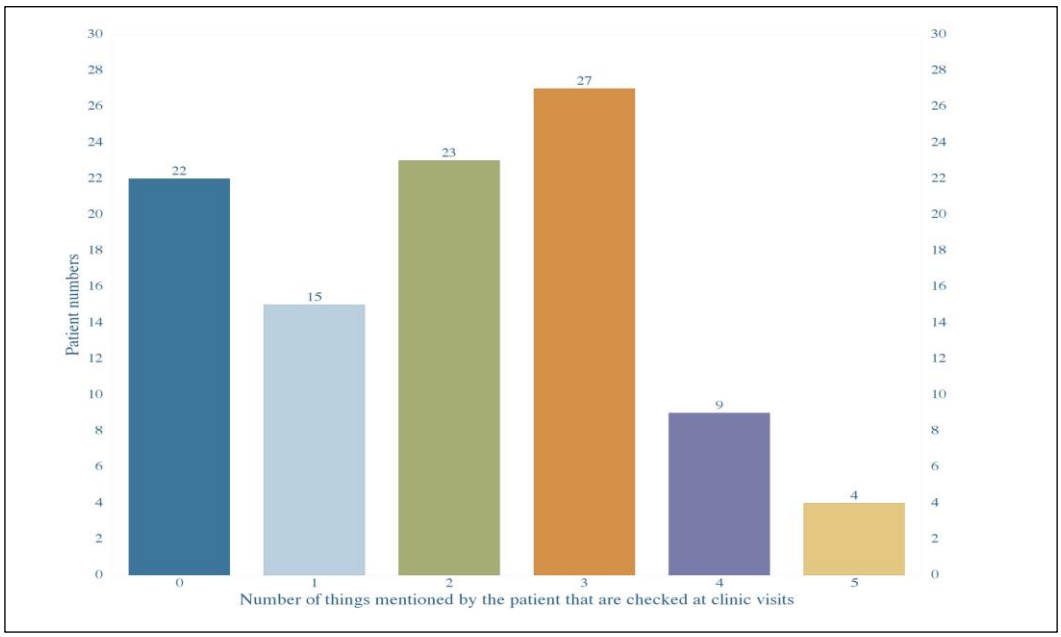

Figure 4: Knowledge of clinic checks versus patient numbers

The knowledge of the patients with respect to things that are measured in the clinic to ensure their well-being was very poor. From the figure above it can be seen than 22 % of the patients could not mention a single clinic check performed to make sure that they are healthy. Items mentioned included the following in decreasing frequency: 61% mentioned glucose, 41% blood pressure, 26% weight, 16% cholesterol, 11% renal function and only 4% mentioned HBA_{1C}.

What was alarming was that the above patients also did not know if their control of these parameters was good or bad. The following table lists the result of this analysis. The values used to decide if the evaluation was correct are listed in the materials and methods section:

Table 18: Diabetes knowledge of specific clinic checks known and evaluation result grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Patients able to mention specific clinic checks					
Blood pressure	n (%)	41 (41%)	21 (37%)	20 (47%)	0.266**
Blood pressure evaluation correct	n (%)	28 (28%)	16 (28%)	12 (28%)	
Blood pressure evaluation wrong	n (%)	13 (13%)	5 (9%)	8 (19%)	
Cholesterol	n (%)	16 (16%)	10 (18%)	6 (14%)	0.738**
Cholesterol evaluation correct	n (%)	6 (6%)	3 (5%)	3 (7%)	
Cholesterol evaluation wrong	n (%)	10 (10%)	7 (12%)	3 (7%)	
Glucose	n (%)	61 (61%)	31 (54%)	30 (70%)	0.490**
Glucose evaluation correct	n (%)	38 (38%)	20 (35%)	18 (42%)	
Glucose evaluation wrong	n (%)	23 (23%)	11 (19%)	12 (28%)	
HBA_{1C}	n (%)	4 (4%)	3 (5%)	1 (2%)	0.248**
HBA_{1C} evaluation correct	n (%)	2 (2%)	1 (2%)	1 (2%)	
HBA_{1C} evaluation wrong	n (%)	2 (2%)	2 (4%)	0 (0%)	
Renal function	n (%)	11 (11%)	5 (9%)	6 (14%)	0.248**
Renal evaluation correct	n (%)	5 (5%)	3 (5%)	2 (5%)	
Renal evaluation wrong	n (%)	6 (6%)	2 (4%)	4 (9%)	
Weight	n (%)	26 (26%)	17 (30%)	9 (21%)	0.641**
Weight evaluation correct	n (%)	17 (17%)	12 (21%)	6 (14%)	
Weight evaluation wrong	n (%)	9 (9%)	5 (9%)	4 (9%)	
Combinations correct					0.313**
Blood pressure, glucose, cholesterol	n (%)	1 (1%)	1 (2%)	0 (0%)	
Blood pressure, glucose	n (%)	18 (18%)	13 (23%)	5 (12%)	
Blood pressure, Cholesterol, HBA_{1C}	n (%)	1 (1%)	1 (2%)	0 (0%)	
Blood pressure, glucose, weight	n (%)	5 (5%)	4 (7%)	1 (2%)	

** Chi-square test

Only 4 patients (4%) could mention that the HBA_{1C} was used to monitor their glucose. Of these only 2 of them (50%) were correct with respect to the status of their HBA_{1C}.

There were very few patients who were able to mention more than one clinic check in combination with a correct assessment of its status, ie low, normal or high. From the above table it is clear that only a single patient could mention blood pressure, cholesterol and HBA_{1C}.

Table 19: Diabetes complications knowledge grouped by HBA_{1C} - macrovascular

Parameter		**All patients (n=100)**	**HBA_{1C} below mean (<= 9.74%) n=57**	**HBA_{1C} above mean (>9.74%) n=43**	**p value**
Macrovascular complications known					
Stroke	n (%)	26 (26%)	12 (21%)	14 (33%)	0.194**
Peripheral vascular disease	n (%)	56 (56%)	31 (54%)	25 (58%)	0.708**
Ischaemic heart disease	n (%)	39 (39%)	18 (32%)	12 (49%)	0.080**
Adding number of macrovascular complications known together					0.166 **
0	n (%)	21 (21%)	13 (23%)	8 (19%)	
1	n (%)	45 (45%)	29 (51%)	16 (37%)	
2	n (%)	26 (26%)	13 (23%)	13 (30%)	
3	n (%)	8 (8%)	2 (4%)	6 (14%)	

** Chi-square test

Table 20: Diabetes complications knowledge grouped by HBA_{1C} - microvascular

Parameter		**All patients (n=100)**	**HBA_{1C} below mean (<= 9.74%) n=57**	**HBA_{1C} above mean (>9.74%) n=43**	**p value**
Microvascular complications known					
Diabetic retinopathy	n (%)	60 (60%)	36 (63%)	24 (56%)	0.458**
Diabetic neuropathy	n (%)	17 (17%)	9 (16%)	8 (19%)	0.711**
Diabetic nephropathy	n (%)	36 (36%)	19 (33%)	17 (40%)	0.522**
Autonomic dysfunction	n (%)	8 (8%)	6 (11%)	2 (5%)	0.284**
Adding number of microvascular complications known together					0.996**
0	n (%)	28 (28%)	11 (19%)	17 (40%)	
1	n (%)	25 (25%)	14 (25%)	11 (26%)	
2	n (%)	33 (33%)	19 (33%)	14 (33%)	
3	n (%)	10 (10%)	6 (11%)	4 (9%)	

** Chi-square test

Table 21: Diabetes complications knowledge grouped by HBA_{1C} - coma

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Diabetic coma complications known					
Diabetic coma – hyperglycemia	n (%)	13 (13%)	9 (16%)	4 (9%)	0.339**
Diabetic coma – hypoglycemia	n (%)	20 (20%)	12 (21%)	8 (19%)	0.762**
Adding number of diabetic coma complications known together					0.713**
0	n (%)	75 (75%)	42 (74%)	33 (77%)	
1	n (%)	17 (17%)	11 (19%)	6 (14%)	
2	n (%)	8 (8%)	5 (9%)	3 (7%)	

** Chi-square test

Table 22: Diabetes complications knowledge grouped by HBA_{1C} - total

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Adding number of macro-, micro- and diabetic coma complications known together (total)					0.467**
0	n (%)	6 (6%)	3 (5%)	3 (7%)	
1	n (%)	9 (9%)	4 (7%)	5 (12%)	
2	n (%)	29 (29%)	19 (33%)	10 (23%)	
3	n (%)	28 (28%)	16 (28%)	12 (28%)	
4	n (%)	20 (20%)	13 (23%)	7 (16%)	
5	n (%)	5 (5%)	2 (4%)	3 (7%)	
6	n (%)	2 (2%)	0 (0%)	2 (5%)	
7	n (%)	1 (1%)	0 (0%)	1 (2%)	

** Chi-square test

Knowledge of diabetic complications, macrovascular (p=0.166), microvascular (p=0.996) and diabetic coma complications (p=0.996) were not statistically significant. Knowledge of ischaemic heart disease (p=0.080) was significant enough but patient numbers in this subgroup were insufficient for the logistic regression model.

Knowledge of diabetes complications was poor in the study population. Twenty one percent could not mention a single macrovascular event, 32% not a single microvascular event and 75% not a single diabetic coma complication.

Table 23: Height for weight evaluation grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
For your height, do you think you weigh too much?					0.400**
No, evaluation correct	n (%)	12 (12%)	9 (16%)	3 (7%)	
No, evaluation wrong	n (%)	37 (37%)	21 (37%)	16 (37%)	

** Chi-square test

The patient's insight with respect to his or her own weight was not found to be a significant predictor of glycaemic control (p=0.400). Interestingly enough, all the patients who answered "yes" to the question: "Do you think you weigh too much for your height?" (n=46) were correct in their assessment. A body mass index of 22-25 kg/m^2 was used as normal for this assessment.

Table 24: Exercise frequency, diabetes education and dietician visit grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Exercise frequency					0.773**
Daily	n (%)	30 (30%)	16 (28%)	14 (33%)	
6 times per week	n (%)	0 (0%)	0 (0%)	0 (0%)	
5 times per week	n (%)	2 (2%)	0 (0%)	2 (5%)	
4 times per week	n (%)	3 (3%)	1 (2%)	2 (5%)	
3 times per week	n (%)	11 (11%)	8 (14%)	3 (7%)	
2 times per week	n (%)	11 (11%)	5 (9%)	6 (14%)	
Once a week	n (%)	3 (3%)	2 (4%)	1 (2%)	
Less often than above	n (%)	21 (21%)	14 (25%)	7 (16%)	
No exercise at all	n (%)	18 (18%)	10 (18%)	8 (19%)	
Not answered	n (%)	1 (1%)	0	1 (2%)	
Did the patient receive diabetes education at Helen Joseph Hospital diabetic clinic?					0.871**
Yes	n (%)	34 (34%)	19 (33%)	15 (35%)	
No	n (%)	66 (66%)	38 (67%)	28 (65%)	
Did the patient see a dietician in the previous year?					0.628**
Yes	n (%)	70 (70%)	41 (72%)	29 (67%)	
No	n (%)	30 (30%)	16 (28%)	14 (33%)	

** Chi-square test

Self-reported exercise frequency was not significant in this study (p=0.773) even though previous studies have shown benefit [68, 70 - 72]. This might be due to error in self-reporting. Exercise type and duration was not questioned and these might be confounders.

Diabetes education (p=0.871) and the input of a dietician in the previous year (p=0.628) were not statistically significant. In previous studies diabetes education and knowledge was cited to be a factor that would improve control [76].

3.1.6. Hypo-and hyperglycaemia

Table 25: Hypo- and hyperglycaemia grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Hypoglycaemia symptoms known or experienced					
Dizziness	n (%)	56 (56%)	31 (54%)	25 (58%)	0.708**
Hunger	n (%)	14 (14%)	7 (12%)	7 (16%)	0.568**
Nausea	n (%)	7 (7%)	4 (7%)	3 (7%)	0.994**
Sweating	n (%)	23 (23%)	12 (21%)	11 (26%)	0.594**
Weakness	n (%)	30 (30%)	17 (30%)	13 (30%)	0.965**
Coma	n (%)	15 (15%)	10 (18%)	5 (12%)	0.412**
Headache	n (%)	7 (7%)	4 (7%)	3 (7%)	0.994**
Confusion	n (%)	24 (24%)	9 (16%)	15 (35%)	0.030**
Shakiness/palpitations	n (%)	36 (36%)	17 (30%)	18 (42%)	0.212**
Knowledge or experience of the above hypoglycaemic symptoms					0.575**
0	n (%)	6 (6%)	4 (7%)	2 (5%)	
1	n (%)	20 (20%)	11 (19%)	9 (21%)	
2	n (%)	38 (38%)	25 (44%)	13 (30%)	
3	n (%)	28 (28%)	15 (26%)	13 (30%)	
4	n (%)	8 (8%)	4 (7%)	4 (9%)	
Hypoglycaemic episodes frequency					0.094**
Monthly	n (%)	64 (66%)	41 (75%)	23 (55%)	
Less than monthly	n (%)	14 (14%)	7 (13%)	7 (17%)	
Never	n (%)	19 (20%)	7 (13%)	12 (28%)	
Hypoglycaemic episodes frequency					0.051**
Less than monthly or monthly	n (%)	78 (14%)	48 (87%)	30 (71%)	
Never	n (%)	19 (20%)	7 (13%)	12 (29%)	
Number of hyperglycaemic symptoms experienced or known					
Coma	n (%)	2 (2%)	2 (4%)	0 (0%)	0.100**
Dizziness	n (%)	24 (24%)	12 (21%)	12 (28%)	0.116**
Fatigue	n (%)	20 (20%)	11 (16%)	9 (21%)	0.226**
Polydipsia	n (%)	18 (18%)	12 (21%)	6 (14%)	0.235**
Polyphagia	n (%)	0 (0%)	0 (0%)	0 (0%)	0
Polyuria	n (%)	3 (3%)	1 (2%)	2 (5%)	0.731**
Knowledge/experience of the above hyperglycaemic symptoms					0.662**
0	n (%)	41 (41%)	23 (40%)	18 (42%)	
1	n (%)	48 (48%)	29 (51%)	19 (44%)	
2	n (%)	10 (10%)	5 (9%)	5 (12%)	
3	n (%)	1 (1%)	0 (0%)	1 (2%)	

** Chi-square test

Hypoglycaemic episode frequency was found to be statistically significant (p=0.094) but knowledge of hypoglycaemic (p=0.575) and hyperglycaemic symptoms (p=0.662) were not.

3.1.7. Glucose monitor and use

Table 26: Glucose monitor and use grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Patient has glucose monitor					0.994**
Yes	n (%)	93 (93%)	53 (93%)	40 (93%)	
No	n (%)	7 (7%0	4 (7%)	3 (7%)	
How often glucose checked?					0.990**
Daily	n (%)	71 (81%)	41 (82%)	30 (79%)	
More than daily	n (%)	17 (19%)	9 (18%)	8 (21%)	

** Chi-square test

Having a glucose monitor (p=0.994) and frequency of glucose checks (p=0.990) did not seem to have statistical significance. This is in contrast to the literature supporting the notion that self-monitoring of glucose improves control [79]. A possible confounder here is that the patients in this study most likely did not know what their glucose control should be. This is based on the poor level of knowledge with respect to diabetes found in the study population. Having a monitor without knowledge of glucose targets would be futile. Knowledge of specific glucose targets was not tested during my interview.

3.1.8. Treatment

Table 27: Treatment grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Identification of prescribed medication					
Correct (100%)	n (%)	78 (78%)	46 (81%)	32 (74%)	0.453**
Incorrect (< 100%)	n (%)	22 (22%)	11 (19%)	11 (26%)	
Concomitant medication					
Metformin					0.330*
Not on metformin	n (%)	26 (26%)	17 (30%)	9 (21%)	0.330**
500 mg BD	n (%)	1 (1%)	0 (0%)	1 (2%)	
Other (i.e. 850 mg/day, 850mg BD, 1g BD)	n (%)	73 (73%)	40 (70%)	33 (77%)	
Simvastatin					0.099**
Not on statin	n (%)	10 (10%)	8 (14%)	2 (5%)	
Simvastatin 20 mg/day	n (%)	62 (62%)	37 (65%)	25 (58%)	
Simvastatin 40 or 80 mg/day	n (%)	28 (28%)	12 (21%)	16 (37%)	
Aspirin					0.079**
Not on aspirin	n (%)	7 (7%)	3 (5%)	4 (9%)	
75 mg/day	n (%)	78 (78%)	49 (86%)	29 (67%)	
150 mg/day	n (%)	15 (15%)	5 (9%)	10 (23%)	
Insulin regimen					0.622**
No insulin	n (%)	6 (6%)	3 (%)	3 (7%)	
Once daily insulin	n (%)	9 (9%)	5 (9%)	4 (9%)	
Twice daily insulin	n (%)	67 (67%)	41 (72%)	26 (60%)	
Basal bolus insulin	n (%)	18 (18%)	8 (14%)	10 (23%)	

*T-Test, ** Chi-square test

Most of the patients (n=78) were able to identify their medication without any errors. This finding was remarkable but not significant with respect to predicting glycaemic control (p=0.453).

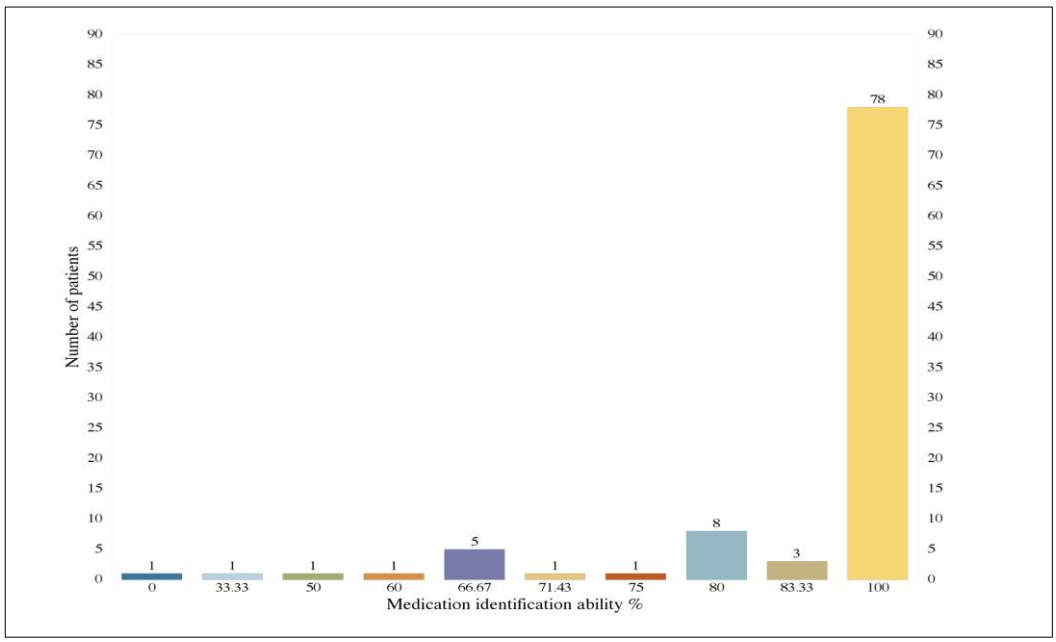

Figure 5: Medication identification ability and number of patients able to achieve it

The dosage of simvastatin (p=0.099) and aspirin (p=0.079) were significant enough to include in the model. Insulin regime (p=0.622) and metformin dosage (p=0.330) did not have an impact on predicting glycaemic control.

The following barriers to taking treatment, in decreasing frequency, were volunteered by the patients: Forget to take medication at times (n=8), nausea secondary to metformin (n=3), difficult to take medication when there is nothing to eat (n=2), difficult to take medication at work due to work schedule (n=2), tired of taking large amounts of medication (n=2), nifedipine gives nightmares (n=1), allergy to aspirin (n=1), confusion between own and mother's medication at times, both have diabetes (n=1), cough secondary to ACE inhibitor (n=1), dizziness secondary to perindopril (n=1), metformin tablet is too big (n=1), sometimes too tired to take medication (n=1).

Seventy-six patients did not report any barrier to taking their medication.

3.1.9. Measurements and examination

Table 28: Measurements grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Body mass index (kg/m^2)	Median (IQR)	32 (28-35)	32 (27-35)	32.3 (29-35)	0.833*
< 25 kg/m^2	n (%)	12 (12%)	6 (21%)	6 (8%)	0.006*
25 – 30 kg/m^2	n (%)	18 (18%)	9 (32%)	9 (13%)	
≥ 30 kg/m^2	n (%)	70 (70%)	13 (47%)	57 (79%)	
Waist circumference (cm)	Median (IQR)	104.5 (97-114)	104 (97-116)	105 (98-113)	0.956^
< 94 cm for males; < 80 cm for females	n (%)	6 (6%)	5 (9%)	1 (2%)	0.179^
≥ 94 cm for males; ≥ 80 cm for females	n (%)	94 (94%)	52 (91%)	42 (98%)	
Neck circumference (cm)	Median (IQR)	37.5 (35-40.5)	37.5 (35-40)	37 (35-40)	0.914^
Systolic blood pressure (mmHg)	Median (IQR)	140.0 (127.5-160.5)	138 (126-156)	145 (132-165)	0.436*
Diastolic blood pressure (mmHg)	Median (IQR)	77 (68-86)	74 (67-80)	81 (74-90)	0.002*
< 70 mmHg	n (%)	26 (26%)	19 (33%)	7 (16%)	0.054*
≥ 80 mmHg	n (%)	38 (38%)	15 (26%)	23 (54%)	0.006*
Glucose on clinic day (mmol/l)	Median (IQR)	10 (7-12)	9.2 (7.1-12)	10.6 (7.3-12.8)	0.493^

*T-Test, ^Kruskal Wallis test, ** Chi-square test

Although both BMI and waist circumference are supported as predictors of glycaemic control in the literature only BMI (p=0.006) was found to be statistically significant in this study [11, 68]. Diastolic blood pressure (p=0.002) was found to be statistically significant but neck circumference (p=0.914), systolic blood pressure (p=0.436) and finger prick glucose measurement on the clinic day (p=0.493) were not significant in predicting glycaemic control.

Table 29: Complications experienced grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Macrovascular complications experienced					
Transient ischaemic attack	n (%)	5 (5%)	4 (7%)	1 (2%)	0.287**
Stroke	n (%)	3 (3%)	2 (4%)	1 (2%)	0.737**
Peripheral vascular disease	n (%)	5 (5%)	3 (5%)	2 (4%)	0.889**
Ischaemic heart disease	n (%)	14 (14%)	6 (11%)	8 (19%)	0.249**
Individual numbers					
0	n (%)	75 (75%)	44 (77%)	31 (72%)	0.304**
1	n (%)	23 (23%)	11 (19%)	12 (28%)	
2	n (%)	2 (2%)	2 (4%)	0 (0%)	
Microvascular complications experienced					
Diabetic retinopathy	n (%)	11 (11%)	6 (11%)	5 (12%)	0.861**
Diabetic neuropathy	n (%)	39 (39%)	22 (39%)	17 (40%)	0.924**
Diabetic nephropathy	n (%)	47 (47%)	29 (51%)	18 (42%)	0.371**
Autonomic dysfunction	n (%)	11 (11%)	8 (14%)	3 (7%)	0.264**
Individual numbers					0.052**
0	n (%)	28 (28%)	11 (19%)	17 (40%)	
1	n (%)	42 (42%)	29 (51%)	13 (30%)	
2	n (%)	24 (24%)	15 (26%)	9 (21%)	
3	n (%)	6 (6%)	2 (4%)	4 (9%)	
Total number of complications experienced (macrovascular + microvascular)					0.04**
0	n (%)	21 (21%)	7 (12%)	14 (33%)	
1	n (%)	42 (42%)	29 (51%)	13 (30%)	
2	n (%)	21 (21%)	14 (25%)	7 (16%)	
3	n (%)	13 (13%)	5 (9%)	8 (19%)	

*T-Test, ^Kruskal Wallis test, ** Chi-square test

The number of microvascular (p=0.052) and total complications (p=0.04) suffered was significant but the number of macrovascular complications suffered (p=0.304) did not show any statistical significance.

3.1.10. Laboratory values

Table 30: Laboratory values grouped by HBA_{1C}

Parameter		All patients (n=100)	HBA_{1C} below mean (<= 9.74%) n=57	HBA_{1C} above mean (>9.74%) n=43	p value
Total cholesterol (mmol/l)	Median (IQR)	4.4 (3.6-5.4)	4.2 (3.6-5.3)	4.7 (3.7-5.7)	0.223^
≥ 4.5 mmol/l	n (%)	50 (50%)	25 (44%)	25 (58%)	0.157^
Triglycerides (mmol/l)	Median (IQR)	1.8 (1.2-2.5)	1.7 (1.2-2.3)	1.9 (1.5-2.9)	0.285^
≥ 1.7 mmol/l	n (%)	57 (57%)	29 (51%)	28 (65%)	0.155^
HDL-C(mmol/l) < 1 mmol/l for males; < 1.2 mmol/l for females	Median (IQR)	1.1 (0.9-1.4)	1.1(0.9-1.4)	1.9 (1.5-2.9)	0.785^
	n (%)	46 (46%)	27 (47%)	19 (44%)	0.752^
LDL-C (mmol/l)	Median (IQR)	2.3 (1.7-3.1)	2.2 (1.6-2.9)	2.6 (1.9-3.3)	0.189^
1.8 – 2.5 mmol/l	n (%)	29 (29%)	18 (33%)	11 (26%)	0.320^
≥ 2.5 mmol/l	n (%)	42 (42%)	20 (37%)	22 (52%)	
Urea (mmol/l)	Median (IQR)	6 (5-8)	6.3 (4.9-7.9)	5.2 (4.5-7.4)	0.133^
≥ 7 mmol/l	n (%)	37 (37%)	24 (42%)	13 (30%)	0.223^
Glomerular filtration rate ($ml/min/1.73m^2$)	Median (IQR)	80 (61-98)	71 (58.5-93)	88 (67-117)	0.0524^
< 60 $ml/min/1.73m^2$	n (%)	25 (25%)	16 (28%)	9 (21%)	0.414^
Urine PCR (g/mmol)	Median (IQR)	0.02 (0.01-0.07)	0.02 (0.01-0.08)	0.03 (0.01-0.06)	0.9293^

^Kruskal Wallis test

eGFR (p=0.0524) were found to be significant enough to include in the logistic regression model.

Total cholesterol (p=0.223), triglycerides (p=0.285), LDL-C (p=0.189), urea (p=0.133), HDL-C (p=0.785) and urine PCR (p=0.9293) were not significant enough to include.

3.2. Logistic regression model results

Variables from the table comparing the group with HBA_{1C} below 9.74 % with the group above were used in a logistic regression model if they had a p value < 0.1. Variables with $p \geq 0.1$ were deemed not to be statistically significant enough and were excluded from further analysis.

The following table lists the result of the logistic regression model. The table lists the crude odds ratio as well as the adjusted odds ratio. The adjusted odds ratio takes all the variables listed in the table into account. Confidence intervals crossing 1.0 make a result less significant as it supports harm and benefit at the same time. If a confidence interval is wide it limits the reliability of a result, if it crosses one no results can be drawn. Larger sample size might influence confidence intervals. All results viewed should bear this in mind.

Even though gender had a p = 0.171 it was also included in the model.

Table 31: Logistic regression analysis of variables with p < 0.1 – demographic analysis

Variable	n (%)	Crude OR (95% CI)	Adjusted OR (95% CI)
Age			
< 50yrs	10 (10%)	1	1
≥ 50 yrs	90 (90%)	0.29 (0.069-1.178)	0.37 (0.06-2.26)
Gender			
Male	28(28%)	1	1
Females	72 (72%)	1.89 (0.75 – 4.73)	2.62 (0.72-9.55)
Duration of diabetes (years)			
≥ 10 yrs	73 (73%)	1	1
< 10 yrs	27 (27%)	1.63 (0.67 – 3.97)	1.36 (0.37-5.02)

Age

If you are over 50, the model predicts that you will have 63% better control compared to a person younger than 50. This is supported by the adjusted odds ratio of 0.37.

Gender

Gender was included in the model even though it had a p value of 0.733 in the table comparing groups with HBA_{1C} above and below the mean. This was done to ensure that gender bias does not confound the final result.

Duration of diabetes

Having diabetes for less than 10 years predicts worse control. The adjusted odds ratio revealed that control would be 1.36 times worse.

Table 32: Logistic regression analysis of variables with $p < 0.1$ – hypoglycaemic frequency

Variable	n (%)	Crude OR (95% CI)	Adjusted OR (95% CI)
Hypoglycaemic episodes frequency			
Never	19 (20%)	1	1
Monthly or Less than monthly	78 (80%)	0.35 (0.12-1)	0.31 (0.09-1.10)

** Statin low: 20 mg Simvastatin daily, Statin high: 40mg or 80mg Simvastatin daily*

Hypoglycaemic episode frequency

Patients with more frequent hypoglycaemic episodes had better control compared to patients who have never had a hypoglycaemic episode. The model revealed a 69% reduction in HBA_{1C} for patients having monthly or fewer hypoglycaemic episodes compared to those who never experienced this phenomenon. Although the confidence interval was narrow, it did cross 1.0. This makes the result less significant.

Table 33: Logistic regression analysis of variables with p < 0.1 – treatment analysis

Variable	n (%)	Crude OR (95% CI)	Adjusted OR (95% CI)
Aspirin dosage			
< 150 mg	85 (85%)	1	1
≥ 150 mg	15 (15%)	3.15 (0.99-10)	6.47 (1.60-26.05)
Simvastatin dosage*			
No statin	10 (10%)	1	1
Statin Low	62 (62%)	2.7 (0.529-13.800)	2.35 (0.31 -18.10)
Statin High	28 (28%)	5.3 (0.954-29.807)	6.97 (0.84 -58.15)

** Statin low: 20 mg Simvastatin daily, Statin high: 40mg or 80mg Simvastatin daily*

Aspirin dosage

Having the patient on an aspirin dosage ≥ 150 mg per day predicted 6.47 times worse control. Although the confidence interval is wide (1.60-26.05), it does not cross 1.0 and therefore makes this finding significant.

Simvastatin dosage

A higher dosage of statin predicted worse glycaemic control. Confidence intervals were wide and crossed 1.0 but 20mg of simvastatin daily predicted 2.35 times worse control, and 40mg to 80mg of simvastatin daily predicted 6.97 times worse control when compared to no simvastatin. This might indicate that patients with poorer control also have more challenging lipid control and in this case would receive a higher dose of statin to achieve adequate lipid control.

Table 34: Logistic regression analysis of variables with p < 0.1 – measurement analysis

Variable	n (%)	Crude OR (95% CI)	Adjusted OR (95% CI)
Body mass index			
<= 25 kg/m^2	12 (12%)	1	1
> 25 kg/m^2	88 (88%)	1.30 (0.72-2.35)	1.09 (0.49-2.41)
Diastolic Blood pressure (mmHg)			
< 70 mmHg	26 (26%)	1	1
≥ 70 mmHg	76 (76%)	2.57 (0.97-6.85)	2.80 (0.80-9.78)

Body mass index

The literature supports weight loss as a measure to improve glycaemic control [11, 68]. This notion is supported by the results in the model. A body mass index of more than 25 kg/m^2 predicted slightly worse control (adjusted odds ratio of 1.09). The confidence interval is unfortunately very wide and crosses 1.0, which reduces the statistical significance.

Diastolic blood pressure

An increase in diastolic blood pressure above 70 predicted 2.80 times worse glycaemic control.

Table 35: Logistic regression analysis of variables with p < 0.1 – laboratory analysis

Variable	n (%)	Crude OR (95% CI)	Adjusted OR (95% CI)
Estimated glomerular filtration rate			
< 60 ml/min/1.73m^2	25 (25%)	1	1
≥ 60 ml/min/1.73m^2	75 (75%)	1.47 (0.58-3.75)	0.90 (0.25-3.27)

Estimated glomerular filtration rate

According to the analysis the crude odds ratio supports better control with a low eGFR and the adjusted odds ratio supports worse control with a low eGFR. The confidence intervals for both the crude and adjusted odds ratios cross 1.0, limiting the statistical significance.

Table 36: Logistic regression analysis of variables with p < 0.1 – complication count

Variable	n (%)	Crude OR (95% CI)	Adjusted OR (95% CI)
Microvascular complication count			
No complication	28 (28%)	1	1
Complications	72 (72%)	0.37 (0.15-0.90)	0.73 (0.11-5.07)
Total complication count			
No complication	21 (22%)	1	1
Complications	76 (78%)	0.29 (0.11-0.81)	0.56 (0.07-4.38)

Microvascular and total complications suffered

It seems that one of the implications of developing a diabetic complication is an improvement in glycaemic control. Suffering any complication improves glycaemic control by 44% according to the crude odds ratio. A microvascular complication suffered improves glycaemic control by 27% compared to no microvascular complication suffered. For both these variables, the confidence interval crossed 1.0, which made the result less significant.

3.3. Logistic regression model with HBA_{1C} of 8.0 % and 7.0% as cut off

An attempt was made to do sensitivity analysis of the logistic regression model by changing the dividing HBA_{1C} value to 8.0 % and 7.0 % respectively.

In this study there were only 20 patients with an $HBA_{1C} \leq 8.0\%$ and 7 patients with a $HBA_{1C} \leq 7.0\%$. Due to the small patient numbers in these groups the models did not reveal any significant result.

3.4. Correlations between HBA_{1C}, blood pressure, lipids, waist circumference and BMI

Correlations were done between HBA_{1C}, blood pressure, lipids, waist circumference and body mass index. The table with the results can be viewed in Appendix E.
The significant correlations with $p < 0.05$ are highlighted in this section.

There was a positive correlation between HBA_{1C} and diastolic blood pressure with an r-value of 0.29981 ($p = 0.0024$). This means for an increase of HBA_{1C} by 1 % the diastolic blood pressure would increase by a factor of 0.29981.

Diastolic blood pressure had 2 significant correlations. The correlation with HBA_{1C} is described above. The second correlation was a positive correlation with the triglyceride level. The r-value was 0.35611 ($p=0.0003$).

There was an expected positive correlation between the body mass index and waist circumference with an r-value of 0.82496 ($p < 0.0001$). There were no other significant correlations with these two variables.

3.5. Comparing diabetic clinic treatment targets, treatment prescribed and laboratory investigation frequency against the 2012 SEMDSA guidelines

3.5.1. Clinic treatment targets against 2012 SEMDSA guidelines

3.5.1.1. Waist circumference

SEMDSA guidelines for waist circumference

The 2012 SEMDSA guideline recommends a waist circumference of less than 80 cm for females and less than 94 cm for males [93].

Waist circumference in the study group

Table 37: Waist circumference of the study group

	Males (n=28)		Females (n=72)	
Waist circumference	**< 94 cm**	**≥ 94 cm**	**< 80 cm**	**≥ 80 cm**
Patient numbers	n=6	n=22	n=0	n=72
Percentage	21%	79%	0%	100%

From the table it is clear that the majority of the patients in this study did not meet the 2012 SEMDSA waist circumference targets for Type 2 diabetes.

3.5.1.2. Blood pressure

2012 SEMDSA guidelines for blood pressure

The 2012 SEMDSA guidelines recommend a systolic blood pressure of 120-140 mmHg and a diastolic blood pressure of 70-80 mmHg [84].

Blood pressures found in the study group

Table 38: Blood pressure in the study group

	Blood pressure lower than guideline blood pressure	Blood pressure meets guideline blood pressure	Blood pressure higher than guideline blood pressure
Total	n=22 (22%)	n=15 (15%)	n=63 (63%)
Receiving hypertension treatment	n=19 (86%)	n=12 (80%)	n=58 (92%)
Not receiving hypertension treatment	n=3 (14%)	n=3 (20%)	n=5 (8%)

Sub analysis of the group of 63 with blood pressures higher than the 2012 SEMDSA guidelines are depicted in the following table:

Table 39: Blood pressure in cohort with SBP ≥ 140 and DBP ≥ 80

Blood pressure group	SBP ≥ 140		DBP ≥ 80
Blood pressure group	**DBP < 80**	**DBP ≥ 80**	**SBP < 140**
Patient numbers	n=25	n=27	n=11
Not receiving hypertension treatment	n=3 (12%)	n=0 (0%)	n=2 (18%)

Blood pressure conclusion

Firstly it must be noted that the above analysis was done on a single clinic blood pressure measurement. This is not a proper way of diagnosing hypertension. It must also be noted that there could be a couple of possible

confounders specific to these measurements. The latest South African hypertension guidelines, published in 2006, specify that the blood pressure must only be taken after the patient has rested for at least 5 minutes. It must be taken with the patient seated and with feet resting on the floor. It also specifies that the blood pressure must not be taken if the patient smoked, drank a caffeine-containing beverage or ate in the previous 30 minutes [91]. Although blood pressures were taken with the patient seated, it was not recorded if any of the other criteria were met. Apart from this, it was also not recorded if the patients took their treatment on the morning of the clinic visit. This in itself might have an impact on the blood pressure reading.

With the above in mind, this single blood pressure measurement used for analysis revealed that 15% of the patients were at blood pressure goal according to the 2012 SEMDSA guideline. This means 85% were not at goal.

86% of the 22 patients with blood pressure lower than the recommended level did receive treatment for hypertension. These patients were over treated for hypertension.

Most of the patients with blood pressure above the recommended level were treated for hypertension. In the subgroup with systolic blood pressure ≥ 140 mmHg and diastolic blood pressure < 80 mmHg only 12% (3/25) were not treated. In the subgroup with systolic blood pressure ≥ 140 mmHg and diastolic blood pressure ≥ 80 mmHg every single patient received

hypertension treatment. In the subgroup with diastolic blood pressure ≥ 80 mmHg and systolic blood pressure <140 mmHg 2/11 (18%) did not receive treatment for hypertension.

3.5.2. Treatment prescribed

3.5.2.1. Aspirin therapy

2012 SEMDSA guidelines for aspirin use

Aspirin (75-300 mg once daily) is indicated for secondary prevention in all patients with established cardiovascular disease. It is recommended as primary prevention for all patients with more than 10% cardiovascular risk over 10 years [94].

Aspirin use in the study group

In the study group of 100 patients, 93 had a prescription for aspirin.

Aspirin not prescribed group

Of the 7 patients found in this group 1 patient reported an allergy to aspirin. The remaining 6 did qualify for aspirin therapy according to the 2012 SEMDSA guidelines. This means 6/7 or 86% of patients who didn't have a prescription for aspirin qualified for aspirin therapy.

Aspirin prescribed group

93 of the 100 patients in the study did receive aspirin. The results are depicted in the following table:

Table 40: Aspirin use in the study group versus SEMDSA guidelines

	Male		Female		
Patient group	**Male <= 50 years**	**Male > 50 years**	**Female <= 60 years**	**Female > 60 years**	**Total**
Patients on aspirin	5	19	27	42	93
Patient should not be on aspirin	5	1	25	0	31
Percentage error	100%	5%	93%	0%	33%

Aspirin conclusion

From the above it can be seen that 86% of the patients who did not have a prescription for aspirin qualified for it.

In the group who did receive aspirin, 33% did not qualify according to the SEMDSA 2012 guidelines [94]. This error was more marked in the younger age group: 100% versus 5% for males and 93% versus 0% for females.

3.5.2.2. Prescription of statins and lipid goals

2012 SEMDSA guidelines for lipids

The 2012 SEMDSA guidelines recommend the following lipid targets:

Total cholesterol: < 4.5 mmol/l

LDL cholesterol: < 1.8 mmol/l (< 2.5 mmol/l if the patient has no cardiovascular disease, no chronic kidney disease, is under 40 years of age or has a duration of diabetes which is less than 10 years and has no other cardiovascular risk factors)

HDL cholesterol: > 1.0 mmol/l (men) and > 1.2 mmol/l (women)

Triglycerides: <1.7 mmol/l [86]

The primary goal is to achieve the LDL-C target and statins are the first line therapy to achieve this. Statins are also indicated for all Type 2 diabetes patients with the following criteria irrespective of LDL-C level:

- Have existing cardiovascular disease
- Have chronic kidney disease (eGFR < 60ml/min/1.73m^2)
- Are older than 40 years of age or have had diabetes for longer than 10 years, with one or more additional cardiovascular risk factor [86] .

Statin use in the study group

In the study group of 100 patients, 90 had a prescription for a statin. Of these, 62 were on 20mg, 27 on 40mg and 1 patient on 80mg of simvastatin. Simvastatin was the only statin prescribed.

Patient group not receiving statins

The 10 patients who did not receive a statin had the following LDL-C values:

Table 41: LDC-C values of patients not on statins

	LDL-C <1.8	1.8 ≤ LDL-C < 2.5	LDL-C ≥ 2.5
Patients numbers	n=4	n=4	n=2

4 patients had a LDL-C value below 1.8mmol/l. By applying the 2012 SEMDSA guidelines all of them qualified for statin therapy except for one. This patient had a LDL-C of 0.7 mmol/l, diabetes duration of 5 years and no additional risk factors for cardiovascular disease. All the patients with LDL-C value >1.8 mmol/l qualified for statin therapy. This means that 9/10 or 90% of the patients who did not receive a statin qualified for it according to the 2012 SEMDSA guidelines.

Statin prescribed group

Ninety patients did receive a statin. It is not possible to fully ascertain if patients in this group were receiving statins in error when compared to guidelines as access to their initial lipogram before initiation of therapy was not available. It is assumed that they would all qualify.

Table 42: LDL-C values of patients on statins

Statin dosage	Patients	LDL-C < 1.8*	1,8 ≤ LDL-C < 2.5*	LDL-C ≥ 2.5*
20mg	62	17	18	27
40mg	27	4	7	16
80mg	1	LDL-C not calculated due to high triglyceride level		

*LDL-C measured in mmol/l

After applying the SEMDSA lipid guidelines with respect to Type 2 diabetes patients in the above table only the patients with a LDL-C < 1.8 mmol/l were at goal. None of the patients in the groups with LDL-C ≥ 1.8 mmol/l were at goal. This means there was not a single patient who met criteria for a less stringent LDL-C goal of < 2.5 mmol/l. If the one patient on 80 mg of simvastatin, who is excluded due to a high triglyceride level, is left from analysis it means that (21/89) 23.5% of the patients were at goal LDL-C level.

Table 43: LDL-C goal versus statin dosage

Statin dosage	LDL-C at goal	LDL-C not at goal
20mg	17/62 = 27%	45/62 = 73%
40mg	4/27 = 15%	23/27 = 85%
80mg	Not calculated due to high TGL value	

Statin prescription and lipid goals conclusion

Ninety percent of the patients who were not receiving statin therapy required it according to the guidelines.

The largest proportion of the group on statins was not controlled at goal: 73% of the group on 20mg and 85% of the group on 40mg were not at goal. In total 23.5% of patients on statins were at goal according to the 2012 SEMDSA guidelines.

3.5.3. Laboratory investigation frequency

3.5.3.1. Urea, creatinine and electrolytes testing frequency

2012 SEMDSA guidelines

The 2012 SEMDSA guidelines state that urea, creatinine and electrolytes (U&E) should be checked annually for patients with normal renal function. It should be checked every 3 months if the eGFR is < 60 ml/min/1.73m^2 or if the albumin creatinine ratio (ACR) is abnormal [92].

Urea, creatinine and electrolyte testing in the study group

All of the patients in the study group did have a previous U&E test on the laboratory computer system. Thirty one of the patients ie 31% did not have a U&E done in the previous year. The mean time between the research clinic visit date and the previous U&E test date was 302.8 days.

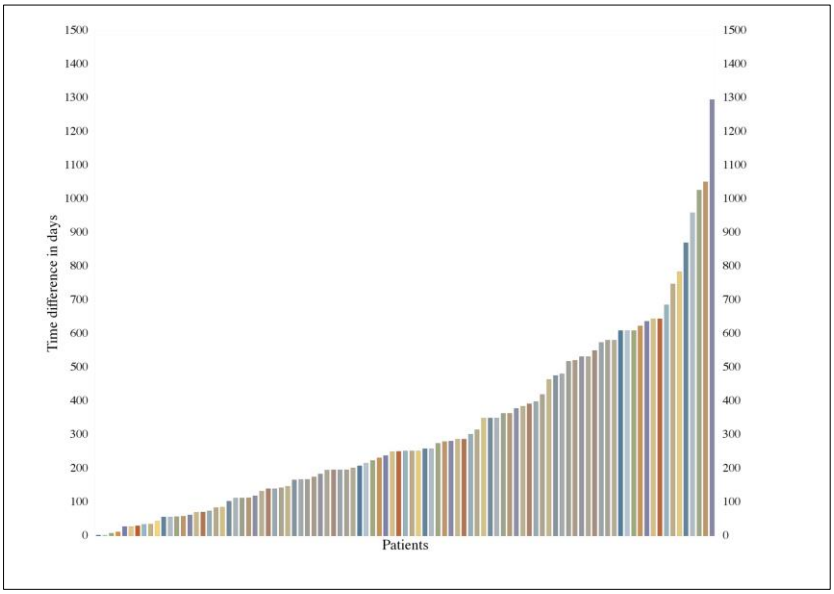

Figure 6: Time difference between research clinic date and previous U&E taken

Patients with eGFR < 60 ml/min/1.73m^2

There were 25 patients with an eGFR < 60 ml/min/1.73m^2.

Table 44: Time of previous U&E before clinic date in group with eGFR < 60 ml/min/1.73m^2

Previous U&E (months)	< 3	3-6	6-12	> 12	Total
Numbers	7 (28%)	7 (28%)	3 (12%)	8 (32%)	25

From this table it is clear that only 7 patients in the group of 25 with eGFR < 60 ml/min/1.73m^2 (28%) met the 2012 SEMDSA guidelines for U&E screening frequency. The mean time between previous U&E date and clinic visit date for this group was 296.28 days.

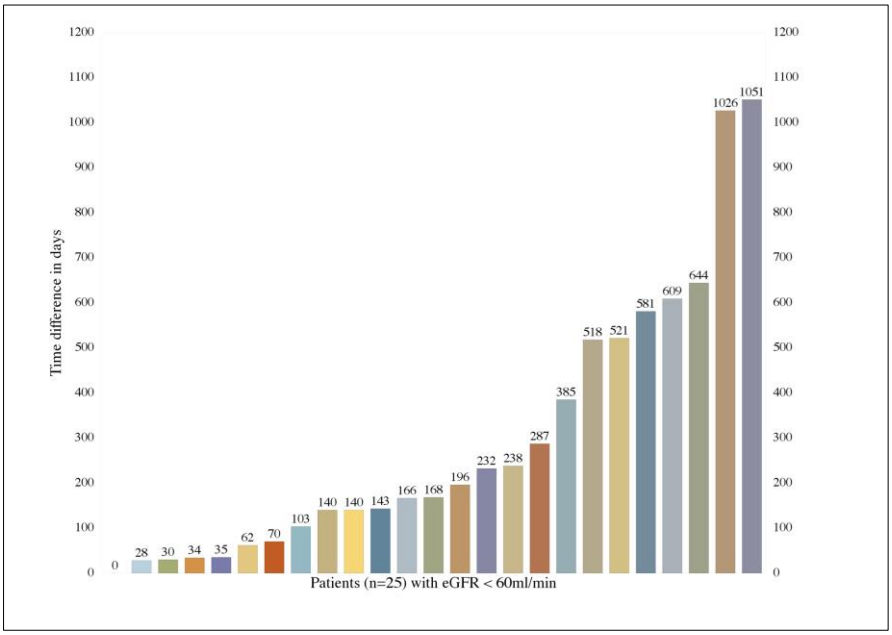

Figure 7: Time between research clinic date and previous U&E done in patients with GFR < 60 ml/min/1.73m^2

Conclusion

The frequency of U&E monitoring in the clinic for this study group does not meet the standard put forward by the SEMDSA guidelines. This also holds for the patients with renal impairment.

3.5.3.2. Lipogram testing frequency

2012 SEMDSA guideline

The SEMDSA guideline for lipid monitoring recommends annual testing. It should be done more often if lipids are high or after treatment has been altered. [86]

Lipogram frequency found in this study

All of the patients in the study did have a lipogram on the laboratory computer system. Thirty seven of the 100 patients (37%) did not have a lipogram listed for the previous year.

The mean time between the clinic study visit date and lipogram date listed on the laboratory system was 378.68 days.

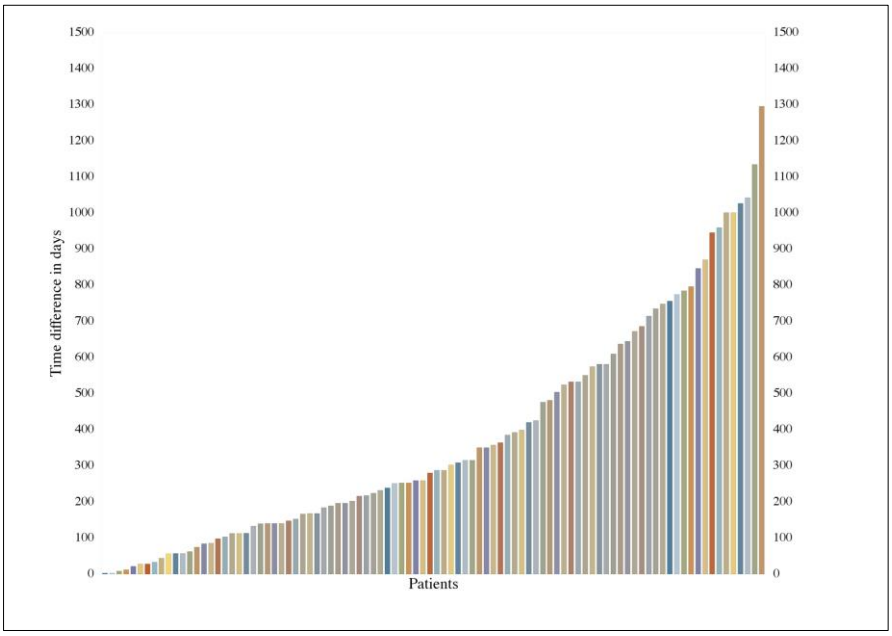

Figure 8: Time difference between research clinic date and lipogram date

Conclusion

The frequency of lipogram monitoring in the clinic for this study group did not meet the standard put forward by the 2012 SEMDSA guidelines.

3.5.3.3. Screening for chronic kidney disease (CKD) using ACR

2012 SEMDSA guidelines

The SEMDSA guidelines suggest screening for CKD with ACR in all Type 2 diabetes patients on presentation. If this initial value is abnormal it should be repeated within 3 months to confirm the diagnosis of CKD. If a diagnosis of CKD is confirmed the patient should be monitored with an ACR test every 6 months [92] .

ACR screening in the total study population

Thirty two (32%) of patients in the study population did not have any urine protein test registered on the National Health Laboratory System (NHLS).

Most patients had a urine PCR test registered (n=47). Unfortunately the urine PCR test is not sensitive enough to reveal microalbuminuria. Only 21 (21%) patients were screened for diabetic nephropathy using an ACR test. The breakdown of the test frequency of the 68 patients (21 with ACR and 47 with PCR) who did have urine proteins done are presented in the next table:

Table 45: Time between clinic date and previous urine protein analysis done date

	Number of patients	Number of tests done in the previous year	Mean time in days between research date and previous test result
Patients with urine ACR	n=21	7 (33%)	802.66
Patients with urine PCR	n=47	9 (19%)	903.5

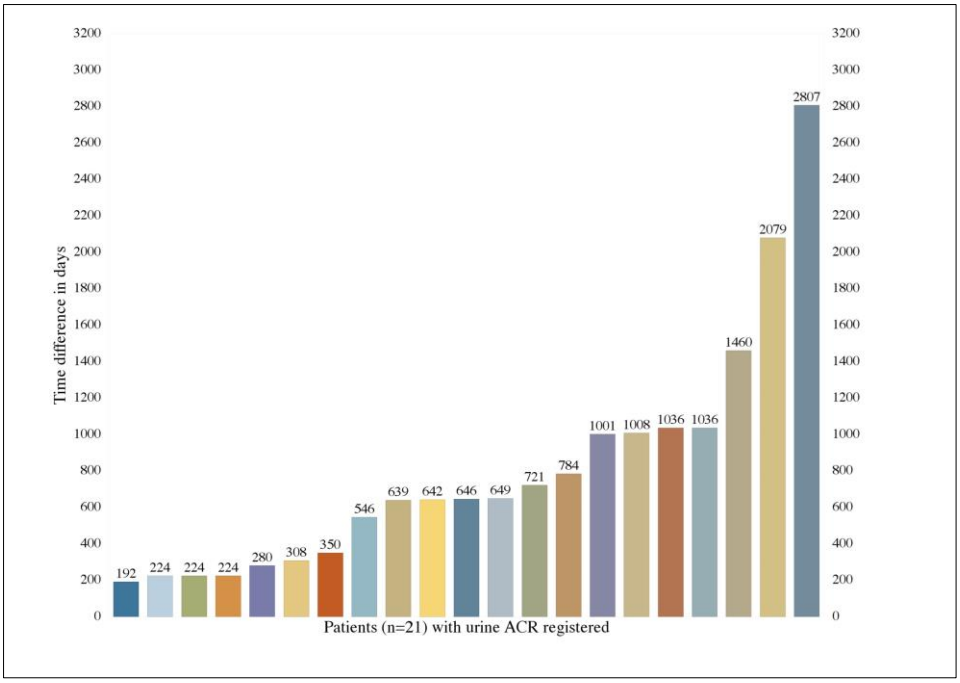

Figure 9: Time difference between research clinic date and previous urine ACR test date

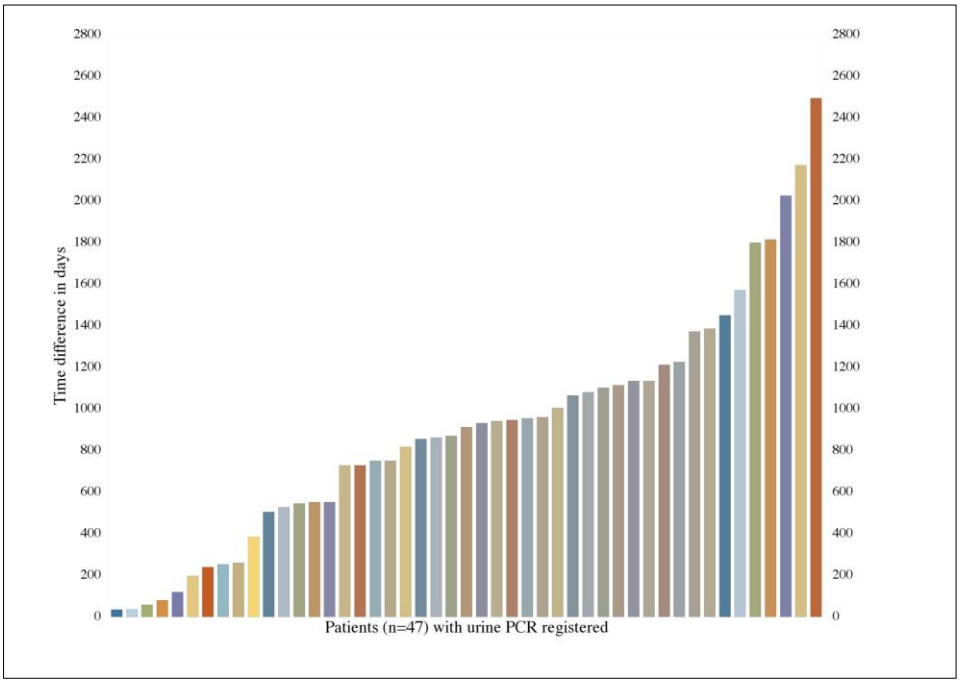

Figure 10: Time difference between research clinic date and previous urine PCR test date

Urine protein screening in the subgroup with eGFR < 60 ml/min/1.73m^2

As noted previously, 25 patients in the study population had an eGFR of less than 60 ml/min/1.73m^2.

Table 46: Urine protein analysis for the 25 patients with eGFR < 60 ml/min/1.73m^2

	Number of patients	Number of tests done in the previous year	Tests done in the last 6 months
Patients with urine ACR	n=0	0 (0%)	0
Patients with urine PCR	n=13	3 (23%)	3 (23%)

2012 SEMDSA guidelines for diagnosis of CKD according to urine ACR

The 2012 SEMDSA guidelines define microalbuminuria in males as a urine ACR > 2.0 mg/mmol. In order to diagnose CKD a single abnormal urine

ACR with the addition of 1 out of 2 abnormal values is needed, when repeating the subsequent tests within 3 months of the initial test [3,84,86] . A single urine ACR test was used for the following analysis:

Urine protein screening in males with ACR > 2.0 mg/mmol

There were 4 males with an ACR > 2.0 mg/mmol. None of them had a urine ACR test listed for the 6 months prior to the research clinic date. Three out of four (75%) of them had a urine ACR test done in the previous year. The mean time between the research clinic date and the previous urine ACR test date for this subset was 693.75 days.

Urine protein screening in females with ACR > 2.8 mg/mmol

As in the previous section a single urine ACR value was used for the following analysis. There were 5 females in the study population with an ACR > 2.8 mg/mmol. In this subgroup of patients none of the previous urine ACR tests were done in the previous 6 months. Two (40%) of them were done in the previous year.

The mean time between the research clinic date and the previous urine ACR test date was 506.8 days.

Conclusion

It seems that most of the patients in this study population (47%) were screened for proteinuria using the incorrect test (urine PCR).

The screening frequency does not meet the 2012 SEMDSA guidelines.

4. CHAPTER 4 – DISCUSSION

4.0. Description of the study population

4.0.1. Smoking

It was heartening to see that 35% of the patients were ex-smokers. This number was higher than the number of smokers in the group (26%) and also higher than the number of non-smokers (31%).

4.0.2. Employment, income and living conditions

Most of the patients in this cohort were not employed (86%) but this includes a large number of retired patients. In the age group < 60 years (n=34), 22 (65%) were not employed which is an alarming unemployment rate. The median income per person for this study population was R1200 per month with an interquartile range of R600 - R1750. There were, therefore, similar incomes across the study participants. This poor monthly income might be a major confounder in this study as healthy living is expensive, especially from a diet perspective. But it must be noted that income on it's own can't be used as proxy for diet. Most of the patients lived in a house (n=77). The population density average per house was 1.5 persons per bedroom. Only 9 patients did not have hot water at home. All of the patients in the study had a fridge and toilet facilities.

4.0.3. Measurements

The mean BMI (32.5 ± 6.4 kg/m^2) found in this study is classified as class I obesity according to the WHO. The mean waist circumference (106.4 ± 15.0 cm) in the

group was markedly increased and significantly more than the target recommended by the 2012 SEMDSA guidelines [86].

The females were more obese compared to the males, especially after removing the largest male individual who had a waist circumference of 106 cm and a BMI of 54.90 kg/m^2 from the male group.

The mean systolic blood pressure of 144.5 mmHg is classified as stage I hypertension according to the JNC VII criteria [85]. The mean diastolic blood pressure of 78.0 mmHg would be classified as acceptable [85].

4.0.4. Laboratory results

The mean clinic glucose for the day, 10.4 ± 4.8 mmol/l, was higher than the post prandial goal of 5.0-10.0 mmol/l stipulated by the 2012 SEMDSA guidelines for the majority of Type 2 diabetic patients [3].

The mean HBA_{1C} for the total group is not close to the 2012 SEMDSA guidelines for control and would constitute poor glycaemic control [3]. The value of 9.74 % is not even close to the less stringent goal of 7.5 % to 8.0 %, the goal recommeded for the elderly, hypoglycaemic unaware, poor short-term prognosis, established cardiovascular disease or high cardiovascular risk patients [3].

The mean HBA_{1C} was lower in the male group (9.19 ± 1.9 %) when compared to the females (9.95 ± 2.27 %). This could be explained by the increased adiposity in the female group signified by increased BMI and waist circumference when

compared to the males. As mentioned previously, removal from analysis of the largest individual in the study group, a male with a waist circumference of 186 cm and BMI of 54.90 kg/m^2, makes the difference in adiposity between the male and female group even more pronounced.

The mean total cholesterol, LDL-C and HDL-C are close to the SEMDSA guidelines [86] . It must be noted that the mean LDL-C value (2.52 mmol/l) was close to goal (< 2.50 mmol/l) for patients who meet all of the following criteria: less than 40 years old or duration of diabetes less than 10 years, no cardiovascular disease, no chronic kidney disease and no other cardiovascular risk factors [86] . Most of the patients in the study population did not fall into this category and would need an LDL-C value < 1.8 mmol/l according to the 2012 SEMDSA guidelines [86] . The mean triglyceride value (2.08 mmol/l) was well above the recommended value of 1.7 mmol/l recommended by SEMDSA [86] . However the laboratory results did not specify if the triglyceride test was done on a fasting blood sample which might impact the level recorded.

Unfortunately there were only 21 patients with a registered urine albumin/creatinine ratio. The mean value for the males (n=6) and females (n=15) were in the microalbuminuria range. The microalbuminuria range is 2.0 – 20.0 mg/mmol for males and 2.8 – 28.0 mg/mmol for females according the 2012 SEMDSA guidelines [92] . Most of the patients with urine protein analysis (n=47) had a urine protein/creatinine ratio listed on the National Health Laboratory System (NHLS). This is the incorrect test for assessment of proteinuria in diabetes, a urine albumin/creatinine ratio test is recommended.

4.1. Results of the statistical analysis

The following section discusses the variables used in this study and their impact on glycaemic control.

4.1.1. Variables found not to have a statistical impact on glycaemic control

Although a paper published in 2006 cited education level as a predictor of glycaemic control this was not supported in our study [71]. It must be noted that the mean education level in this group was low and that this might be the confounding factor. Comparing 2 groups with marked differences in education level might reveal a different result.

The duration of diabetic clinic attendance did not reveal a statistical improvement in glycaemic control.

The number of co-morbidities suffered by a patient, even after sub-analysis of the most prevalent ones, did not predict glycaemic control.

The patient's opinion about the seriousness of diabetes as a disease did not have any bearing on their level of glycaemic control. Smoking is cited to worsen glycaemic control by increasing insulin resistance [18]. In this study no association could be found between smoking and glycaemic control. None of the smoking variables (being a smoker, non-smoker, previous smoker, the pack years smoked or duration stopped smoking for previous smokers) had any statistical bearing on glycaemic control.

In this study, the mean income per person did not show any effect with respect to glycaemic control. Although proper nutrition is listed in the literature as a factor improving glycaemic control, it is expensive [78]. Most of the patients in the study had similar income. Comparing this group of patients to a group with substantially higher income might have shown an effect on glycaemic control.

The patient's opinion about his or her own health did not have a statistical impact on glycaemic control. It was surprising to find that neither knowledge of ways to improve glucose levels or knowledge of things checked in the clinic played any role with respect to glycaemic control as diabetes education, which would improve these entities, has been shown to improve glycaemic control [76].

The average knowledge of diabetes was very poor. This includes ways to improve glucose, things to check in the clinic as well as possible complications. Only 2 patients (2%) were able to mention 4 things that would improve your glucose control. Six patients could not mention anything that would improve their glycaemic control. An alarming 22% of patients could not mention a single test or procedure done in the clinic to ensure their well-being as a diabetic patient. Exercise has been cited as improving glucose control [68]. This was not found in this study. Exercise frequency reported did not have an impact on glycaemic control. This finding could be secondary to self-reporting bias.

Unfortunately this study could not show any benefit related to diabetes education even though this is supported in the literature [76]. The input from a dietician in the previous year also had no effect on glycaemic control.

Internet access did not have any impact on glycaemic control.

Knowledge or experience of hypo- and hyperglycaemic symptoms did not statistically predict glycaemic control. The literature cites that self-monitoring of blood glucose improves control [79]. Neither the ownership of a glucose monitor or the frequency of glucose testing revealed any benefit with respect to glycaemic control. This could be explained by the poor knowledge base found amongst the study population. Having a monitor without the knowledge of what to aim for and how to get there would be futile.

Glucose measurement value on the research clinic day as well as the value did not statistically predict glycaemic control.

The finding that 78% of the patients were able to identify their medication with 100% accuracy was surprising. Although this finding does not imply compliance it at least confirms that the diabetic population included in this study know their medication and how to take it. The correct identification of medicine did not, however, translate to glycaemic control.

Neck circumference and systolic blood pressure had no statistically significant influence on glycaemic control.

Neither the total cholesterol, LDL-C, HDL-C, triglyceride nor urea value had any statistically significant effect on glycaemic control. The urine PCR value also did not emerge as a predictor of glycaemic control.

4.1.2. Variables with an impact on glycaemic control

4.1.2.1. Factors associated with improved glycaemic control

Increase in age, increase in duration of diabetes, increase in eGFR, more frequent hypoglycaemic episodes and suffering a diabetic complication, specifically a microvascular complication, all seem to be associated with better glycaemic control.

In comparing the above list with the current available literature only an increase in age is cited [77].

An increase in age and duration of diabetes might both lead to a decrease in glomerular filtration rate as the kidney is part of insulin metabolism [95]. With long standing diabetes the incidence of microvascular complications, in this case renal dysfunction, would be increased. This is in contrast to the finding in this study that an increase in eGFR was associated with better glycaemic control. However it must be stated that the confidence intervals for all these variables did cross 1.0, making the results less reliable.

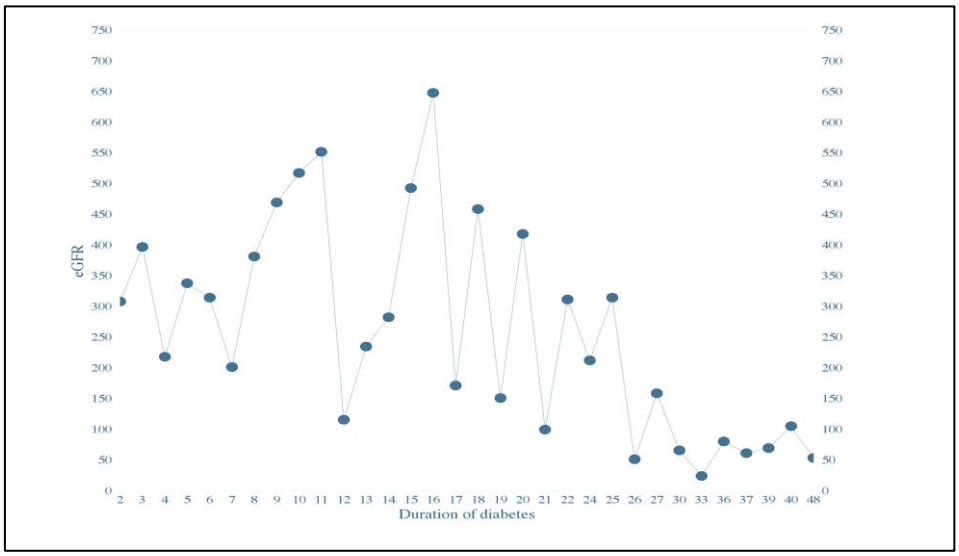

Figure 11: Duration of diabetes versus eGFR in study population

One could postulate that knowledge of diabetes should increase with duration of diabetes culminating in an improvement of control. In this study longer duration of diabetes revealed a poorer knowledge of diabetes as can be seen in the following figure:

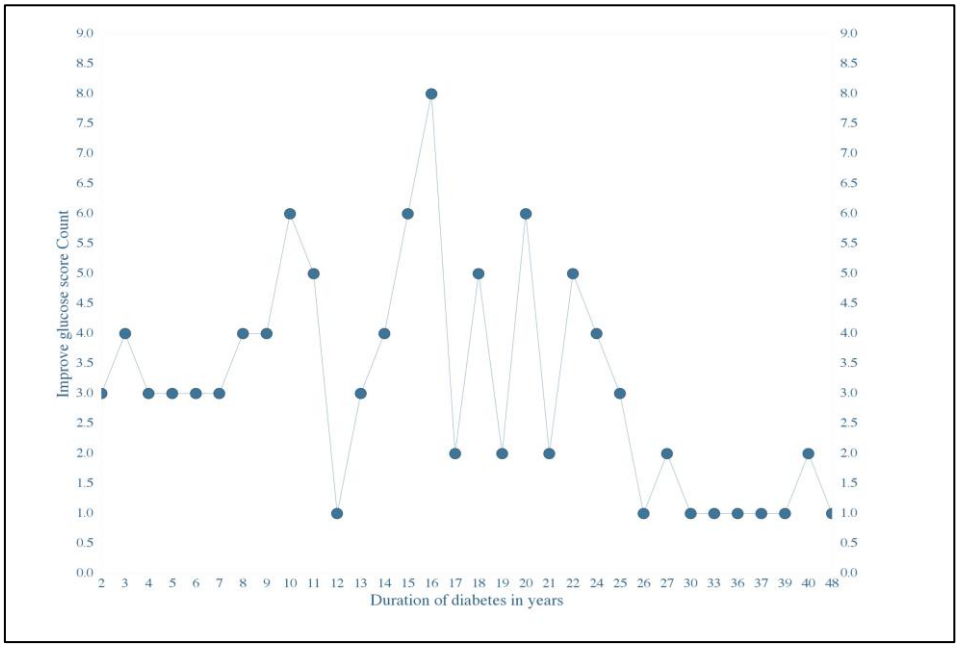

Figure 12: Duration of diabetes versus knowledge to improve control in study population

More frequent hypoglycaemic episodes might indicate patients actually attempting control and thus achieving better values. Analysis of this

variable was done by contrasting these patients with patients who have never experienced any symptoms of hypoglycaemia.

The finding that experience of a diabetic complication improves control might show that real experience speaks louder than words.

4.1.2.2. Factors associated with poorer glycaemic control

An increase in diastolic blood pressure, a higher dose of daily aspirin, a higher dose of simvastatin and an increased body mass index were all associated with worse glycaemic control.

None of the above, except for body mass index has been cited in the literature reviewed for this research [11].

It was interesting to find that an increased diastolic blood pressure was a predictor of poorer glycaemic control. This might be a surrogate marker for worsening autonomic dysfunction with impact on glycaemic control.

It must be noted that the confidence interval found with the increased aspirin dose variable did not cross 1.0. This makes this finding highly significant. Although compliance was not measured, most patients identified their medication accurately. Seventy-eight percent made no error with medication identification, which would support excellent compliance. Perhaps aspirin in itself has pharmacodynamic properties with an impact on glycaemic control. This could be a topic for further research.

A number of articles have reported an increased risk of developing diabetes with the use of statins [20,22,96]. The effect of statins on glycaemic control in known diabetes is less well known. Although this was an observational study and confidence intervals were wide and crossing 1.0, the statin dosage analysis result revealed that higher statin dosages was associated with poorer glycaemic control.

4.2. Correlations between HBA_{1C} and blood pressure, lipids, waist circumference and body mass index

Most of the correlations found in this study were expected. Positive correlations between total cholesterol, LDL-C and HDL-C, would be expected, as would a positive correlation between systolic and diastolic blood pressure and between body mass index and waist circumference.

It was interesting to note that diastolic blood pressure did not only have a positive correlation with the systolic blood pressure but also with the HBA_{1C} and triglyceride levels. The HBA_{1C} correlation might be related to the microvascular complication of autonomic dysfunction as diastolic blood pressure is related to the autonomic function of the vasculature. The other possibility is that glucose might have a direct effect on vascular tone as well as the production of triglycerides.

Body mass index and waist circumference did not have a significant correlation with HBA_{1C} in this study ($p = 0.8152$ and 0.7639 respectively). This was a surprising finding. Obesity has been cited as a risk factor for developing diabetes and weight loss has been cited to improve glycaemic control [11].

4.3. Comparison of diabetic clinic treatment targets, treatment prescribed and laboratory investigation against the 2012 SEMDSA guidelines

Very few patients met the targets for waist circumference and blood pressure in this study when compared to the 2012 SEMDSA guidelines. Only 6 males (21%) and no females had a waist circumference at target.

Blood pressure analysis was done on a single clinic blood pressure measurement. Previously it was mentioned that there might be possible confounders to the blood pressure measurement (Section 3.5.1.2). If the blood pressure reading can be trusted only 15 patients (15%) were at goal blood pressure according to the 2012 SEMDSA guidelines. A worrying 86% of the patients with blood pressure below the guideline blood pressure were receiving hypertensive treatment. Most of the patients with a blood pressure higher than recommended by the guideline were receiving treatment. Only 5/63 (8%) of patients in this group did not receive anti-hypertensive treatment.

In the group of patients without a prescription for aspirin (n=7), 6 (86%) did qualify according to the 2012 SEMDSA guidelines.

After evaluation of the patients with aspirin prescriptions it was clear that aspirin therapy was over prescribed in the younger male and female population. None of the males ≤ 50 years of age qualified for aspirin therapy but were all receiving it. In the female group ≤ 60 years of age, 25/27 also received aspirin incorrectly according to the 2012 SEMDSA guidelines. The numbers were a lot better in the older patient groups. In the group of males older than 50 only 5% received aspirin in error and in the female group 0%.

In the group of 10 patients who did not have a prescription for a statin, 9 were eligible according to the 2012 SEMDSA guidelines.

Using the latest lipogram result only 23.5% of the patients on statin therapy were at LDL-C goal according to the 2012 SEMDSA guidelines.

The mean time between the research clinic visit date and the previous urea, creatinine and electrolytes measurements recorded on the system was 302.8 days. Thirty one percent of patients did not have a urea, creatinine and electrolyte test performed in the previous year. In the subgroup of patients with renal impairment (n=25), only 7/25 (28%) had a urea, creatinine and electrolyte test listed in the previous 3 months. The frequency of U&E monitoring could be markedly improved in the clinic when compared to the 2012 SEMDSA guidelines.

The mean time between the research clinic date visit and the previous lipogram on the NHLS computer system was 378.68 days. There was no lipogram recorded for 37% of the patients in the previous year. The frequency of lipogram monitoring fell short of the 2012 SEMDSA guidelines and could be improved.

Although the mean time period of clinic attendance for the study population was 10.93 years, there were 32 patients (32%) without any urine protein recorded in the NHLS computer system. For most patients in the study group who did have a urine protein test recorded the wrong investigation in the form of a urine PCR test was requested. A urine PCR test was requested for 47% of the patients. In the urine PCR test group only 19% of tests were conducted in the previous year.

Only 21 patients (21%) were screened for albuminuria using a urine ACR test. In this group only 33% of tests were done in the previous year.

In the subgroup of patients with an eGFR < than 60 ml/min/1.73m^2 none had a listed urine ACR test and 13 had a listed urine PCR test. Of the 13 who had urine PCR tests listed only 3 (23%) were done in the previous year.

In the group of male patients with ACR > 2.0 mg/mmol none of them had a previous ACR done in the 6 months prior to the research clinic date.

In the group of female patients with a urine ACR > 2.8 mg/mmol there were also no urine ACR values for the previous 6 months.

Screening for and monitoring of chronic kidney disease was significantly below standard when compared to the 2012 SEMDSA guidelines.

4.4. Limitations of the study

Some variables cited in the literature were not investigated in this study.

Diet has been cited to have an impact on glycaemic control but was left from data acquisition as proper investigation of this entity would be very time consuming [68].
A recent study published this year found that diabetes was an independent risk factor for dementia [97]. The cohort I examined had significant exposure to diabetes (mean duration of 15.8 years) and was elderly (mean age of 62.8 years). Although I did not screen for

underlying dementia, its presence would have a significant impact on glycaemic control and might have confounded some of the variables.

Adherence to lifestyle modification and medication play a role in glycaemic control. Although medication identification was evaluated, neither compliance with medication or lifestyle adherence was assessed which limits this study.

Using a larger sample size would improve confidence intervals and p values in this study.

It must be kept in mind that the likelihood of finding a chance statistically significant effect exists given the multiple comparisons and the use of non-validated questionnaires and scores.

4.5. Conclusion

It was found to be extremely challenging to narrow down predictors of glycaemic control as a vast number of variables could play a role. To improve statistical significance large patient numbers would be needed to show differences.

In this study a selection of variables were chosen and investigated.
The study population was elderly, had a long duration of diabetes, poor education level and also had very poor socio-economic status.

The knowledge of diabetes management and complications was extremely poor but surprisingly the patients were very knowledgeable with respect to their medication.

Increase in age, duration of diabetes, increase in eGFR , diabetic complications suffered and more frequent hypoglycaemic episodes were all found to be associated with better glycaemic control.

An increased diastolic blood pressure, aspirin dose, statin dose and body mass index all predicted worse glycaemic control.

It was interesting to find that smoking, education level, monthly income and knowledge of diabetes did not predict glycaemic control. As previously stated, the education level and income of the group was similar. Without adequate differences it would be difficult to show statistical effect.

This was an observational study with limited power. Further study would be needed with respect to aspirin and statin dosage and their relationship with glycaemic control.

5. REFERENCES

1. Hu FB. Globalization of diabetes: the role of diet, lifestyle, and genes. Diabetes Care 2011;34:1249-57.
2. Evans MM. Evidence-based practice protocol to improve glucose control in individuals with type 2 diabetes mellitus. Medsurg Nurs 2010;19:317-22.
3. Anonymous Assessment of glycaemic control. Journal of Endocrinology, Metabolism & Diabetes of South Africa 2012;17:S20-2.
4. Paulweber B, Valensi P, Lindström J, et al. A European evidence-based guideline for the prevention of type 2 diabetes. Horm Metab Res 2010;42 Suppl 1:S3-S36.
5. Heideman WH, Nierkens V, Stronks K, et al. DiAlert: a lifestyle education programme aimed at people with a positive family history of type 2 diabetes and overweight, study protocol of a randomised controlled trial. BMC Public Health 2011;11:751.
6. Siitonen N, Pulkkinen L, Lindström J, et al. Association of ADIPOQ gene variants with body weight, type 2 diabetes and serum adiponectin concentrations: the Finnish Diabetes Prevention Study. BMC Med Genet 2011;12:5-.
7. Salopuro T, Pulkkinen L, Lindström J, et al. Genetic variation in leptin receptor gene is associated with type 2 diabetes and body weight: The Finnish Diabetes Prevention Study. Int J Obes (Lond) 2005;29:1245-51.
8. Yates T, Davies MJ, Gray LJ, et al. Levels of physical activity and relationship with markers of diabetes and cardiovascular disease risk in 5474 white European and South Asian adults screened for type 2 diabetes. Prev Med 2010;51:290-4.
9. Qin L, Knol MJ, Corpeleijn E, Stolk RP. Does physical activity modify the risk of obesity for type 2 diabetes: a review of epidemiological data. Eur J Epidemiol 2010;25:5-12.
10. Shi L, Morrison JA, Wiecha J, Horton M, Hayman LL. Healthy lifestyle factors associated with reduced cardiometabolic risk. Br J Nutr 2011;105:747-54.
11. Gill RS, Sharma AM, Gill SS, Birch DW, Karmali S. The impact of obesity on diabetes mellitus and the role of bariatric surgery. Maturitas 2011;69:137-40.
12. Friedl KE. Waist circumference threshold values for type 2 diabetes risk. J Diabetes Sci Technol 2009;3:761-9.
13. Kong W, Langlois M, Kamga-Ngandé C, Gagnon C, Brown C, Baillargeon J. Predictors of success to weight-loss intervention program in individuals at high risk for type 2 diabetes. Diabetes Res Clin Pract 2010;90:147-53.
14. Alberti KGMM, Zimmet P, Shaw J. International Diabetes Federation: a consensus on Type 2 diabetes prevention. Diabet Med 2007;24:451-63.

15. Nilsen V, Bakke PS, Gallefoss F. Effects of lifestyle intervention in persons at risk for type 2 diabetes mellitus - results from a randomised, controlled trial. BMC Public Health 2011;11:893-.
16. Pan A, Sun Q, Bernstein AM, et al. Red meat consumption and risk of type 2 diabetes: 3 cohorts of US adults and an updated meta-analysis. Am J Clin Nutr 2011;94:1088-96.
17. Similä M,E., Valsta LM, Kontto JP, Albanes D, Virtamo J. Low-, medium- and high-glycaemic index carbohydrates and risk of type 2 diabetes in men. Br J Nutr 2011;105:1258-64.
18. Tonstad S. Cigarette smoking, smoking cessation, and diabetes. Diabetes Res Clin Pract 2009;85:4-13.
19. Hur NW, Kim HC, Nam CM, Jee SH, Lee HC, Suh I. Smoking cessation and risk of type 2 diabetes mellitus: Korea Medical Insurance Corporation Study. Eur J Cardiovasc Prev Rehabil 2007;14:244-9.
20. Cannon CP. Balancing the benefits of statins versus a new risk—diabetes. The Lancet 2010;375:700-1.
21. Dagogo-Jack S. HIV Therapy and Diabetes Risk. Diabetes Care June 2008;31:1267-8.
22. Sattar N, Preiss D, Murray HM, et al. Statins and risk of incident diabetes: a collaborative meta-analysis of randomised statin trials. The Lancet 2010;375:735-42.
23. Schwarz EB, Brown JS, Creasman JM, et al. Lactation and maternal risk of type 2 diabetes: a population-based study. Am J Med 2010;123:863.e1,863.e6.
24. Hussain A, Claussen B, Ramachandran A, Williams R. Prevention of type 2 diabetes: a review. Diabetes Res Clin Pract 2007;76:317-26.
25. Anonymous Diagnosis of diabetes mellitus and other disorders of glycaemia, and screening for diabetes mellitus. Journal of Endocrinology, Metabolism & Diabetes of South Africa 2012;17:S7-9.
26. Ackermann RT, Cheng YJ, Williamson DF, Gregg EW. Identifying adults at high risk for diabetes and cardiovascular disease using hemoglobin A1c National Health and Nutrition Examination Survey 2005-2006. Am J Prev Med 2011;40:11-7.
27. Pajunen P, Peltonen M, Eriksson JG, et al. HbA(1c) in diagnosing and predicting Type 2 diabetes in impaired glucose tolerance: the Finnish Diabetes Prevention Study. Diabet Med 2011;28:36-42.
28. Signorello LB, Schlundt DG, Cohen SS, et al. Comparing diabetes prevalence between African Americans and Whites of similar socioeconomic status. Am J Public Health 2007;97:2260-7.
29. Lindqvist PG, Olsson H, Landin-Olsson M. Are active sun exposure habits related to lowering risk of type 2 diabetes mellitus in women, a prospective cohort study? Diabetes Res Clin Pract 2010;90:109-14.
30. Kim C, Park J, Lee K, Kim J, Kim H. Fatty liver is an independent risk factor for the development of Type 2 diabetes in Korean adults. Diabet Med 2008;25:476-81.
31. Norris SL, Zhang X, Avenell A, et al. Long-term effectiveness of weight-loss interventions in adults with pre-diabetes: a review. Am J Prev Med 2005;28:126-39.

32. Viswanathan M, Snehalatha C, Viswanathan V, Vidyavathi P, Indu J, Ramachandran A. Reduction in body weight helps to delay the onset of diabetes even in non-obese with strong family history of the disease. Diabetes Res Clin Pract 1997;35:107-12.
33. Lloret-Linares C, Greenfield JR, Czernichow S. Effect of weight-reducing agents on glycaemic parameters and progression to Type 2 diabetes: a review. Diabet Med 2008;25:1142-50.
34. Margolis KL, Wei F, de Boer I,H., et al. A diet high in low-fat dairy products lowers diabetes risk in postmenopausal women. J Nutr 2011;141:1969-74.
35. Malik VS, Sun Q, van Dam R,M., et al. Adolescent dairy product consumption and risk of type 2 diabetes in middle-aged women. Am J Clin Nutr 2011;94:854-61.
36. Nanri A, Mizoue T, Noda M, et al. Fish intake and type 2 diabetes in Japanese men and women: the Japan Public Health Center-based Prospective Study. Am J Clin Nutr 2011;94:884-91.
37. Snowdon DA, Phillips RL. Does a vegetarian diet reduce the occurrence of diabetes? Am J Public Health 1985;75:507-12.
38. Montonen J, Järvinen R, Heliövaara M, Reunanen A, Aromaa A, Knekt P. Food consumption and the incidence of type II diabetes mellitus. Eur J Clin Nutr 2005;59:441-8.
39. Lee HJ, Kim HC, Vitek L, Nam CM. Algae consumption and risk of type 2 diabetes: Korean National Health and Nutrition Examination Survey in 2005. J Nutr Sci Vitaminol (Tokyo) 2010;56:13-8.
40. de Munter J,S.L., Hu FB, Spiegelman D, Franz M, van Dam R,M. Whole grain, bran, and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review. PLoS Med 2007;4:e261-.
41. Boggs DA, Rosenberg L, Ruiz-Narvaez E, Palmer JR. Coffee, tea, and alcohol intake in relation to risk of type 2 diabetes in African American women. Am J Clin Nutr 2010;92:960-6.
42. Agardh EE, Carlsson S, Ahlbom A, et al. Coffee consumption, type 2 diabetes and impaired glucose tolerance in Swedish men and women. J Intern Med 2004;255:645-52.
43. van Dam R,M. Coffee consumption and risk of type 2 diabetes, cardiovascular diseases, and cancer. Appl Physiol Nutr Metab 2008;33:1269-83.
44. Jing Y, Han G, Hu Y, Bi Y, Li L, Zhu D. Tea consumption and risk of type 2 diabetes: a meta-analysis of cohort studies. J Gen Intern Med 2009;24:557-62.
45. Phung OJ, Sood NA, Sill BE, Coleman CI. Oral anti-diabetic drugs for the prevention of Type 2 diabetes. Diabet Med 2011;28:948-64.
46. Retnakaran R, Qi Y, Harris SB, Hanley AJ, Zinman B. Changes over time in glycemic control, insulin sensitivity, and beta-cell function in response to low-dose metformin and thiazolidinedione combination therapy in patients with impaired glucose tolerance. Diabetes Care 2011;34:1601-4.
47. Mathur G, Noronha B, Rodrigues E, Davis G. The role of angiotensin II type 1 receptor blockers in the prevention and management of diabetes mellitus. Diabetes Obes Metab 2007;9:617-29.

48. Anonymous Reduction in the Incidence of Type 2 Diabetes with Lifestyle Intervention or Metformin. N Engl J Med 2002;346:393-403.
49. Eriksson JG, Lehtovirta M, Ehrnström B, Salmela S, Groop L. Long-term beneficial effects of glipizide treatment on glucose tolerance in subjects with impaired glucose tolerance. J Intern Med 2006;259:553-60.
50. Osei K, Rhinesmith S, Gaillard T, Schuster D. Beneficial metabolic effects of chronic glipizide in obese African Americans with impaired glucose tolerance: implications for primary prevention of type 2 diabetes. Metabolism 2004;53:414-22.
51. Abuissa H, Jones PG, Marso SP, O'Keefe JH, Jr. Angiotensin-converting enzyme inhibitors or angiotensin receptor blockers for prevention of type 2 diabetes: a meta-analysis of randomized clinical trials. J Am Coll Cardiol 2005;46:821-6.
52. Mitri J, Dawson-Hughes B, Hu FB, Pittas AG. Effects of vitamin D and calcium supplementation on pancreatic β cell function, insulin sensitivity, and glycemia in adults at high risk of diabetes: the Calcium and Vitamin D for Diabetes Mellitus (CaDDM) randomized controlled trial. Am J Clin Nutr 2011;94:486-94.
53. Bili A, Sartorius JA, Kirchner HL, et al. Hydroxychloroquine use and decreased risk of diabetes in rheumatoid arthritis patients. J Clin Rheumatol 2011;17:115-20.
54. Beulens JWJ, van der A D,L., Grobbee DE, Sluijs I, Spijkerman AMW, van der Schouw Y,T. Dietary phylloquinone and menaquinones intakes and risk of type 2 diabetes. Diabetes Care 2010;33:1699-705.
55. Villegas R, Gao Y, Dai Q, et al. Dietary calcium and magnesium intakes and the risk of type 2 diabetes: the Shanghai Women's Health Study. Am J Clin Nutr 2009;89:1059-67.
56. de lC, Messiah SE, Arheart KL, Lopez-Mitnik G, Lipshultz SE, Livingstone A. Bariatric surgery significantly decreases the prevalence of type 2 diabetes mellitus and pre-diabetes among morbidly obese multiethnic adults: long-term results. J Am Coll Surg 2011;212:505-11.
57. Gan SSH, Talbot ML, Jorgensen JO. Efficacy of surgery in the management of obesity-related type 2 diabetes mellitus. ANZ J Surg 2007;77:958-62.
58. Baker MK, Simpson K, Lloyd B, Bauman AE, Singh MAF. Behavioral strategies in diabetes prevention programs: a systematic review of randomized controlled trials. Diabetes Res Clin Pract 2011;91:1-12.
59. Yamaoka K, Tango T. Efficacy of lifestyle education to prevent type 2 diabetes: a meta-analysis of randomized controlled trials. Diabetes Care 2005;28:2780-6.
60. Lindström J, Eriksson JG, Valle TT, et al. Prevention of diabetes mellitus in subjects with impaired glucose tolerance in the Finnish Diabetes Prevention Study: results from a randomized clinical trial. J Am Soc Nephrol 2003;14:S108-13.
61. Arase Y, Suzuki F, Suzuki Y, et al. Sustained virological response reduces incidence of onset of type 2 diabetes in chronic hepatitis C. Hepatology 2009;49:739-44.

62. Lindblad U, Lindberg G, Månsson N, et al. Can sulphonylurea addition to lifestyle changes help to delay diabetes development in subjects with impaired fasting glucose? The Nepi ANtidiabetes StudY (NANSY). Diabetes Obes Metab 2011;13:185-8.
63. Nanri A, Mizoue T, Takahashi Y, et al. Soy product and isoflavone intakes are associated with a lower risk of type 2 diabetes in overweight Japanese women. J Nutr 2010;140:580-6.
64. Davila EP, Florez H, Trepka MJ, et al. Long work hours is associated with suboptimal glycemic control among US workers with diabetes. Am J Ind Med 2011;54:375-83.
65. Sullivan LV, Hicks P, Salazar G, Robinson CK. Patient beliefs and sense of control among Spanish-speaking patients with diabetes in northeast Colorado. J Immigr Minor Health 2010;12:384-9.
66. Mackenzie T, Brooks B, O'Connor G. Beverage intake, diabetes, and glucose control of adults in America. Ann Epidemiol 2006;16:688-91.
67. Ciechanowski PS, Katon WJ, Russo JE, Hirsch IB. The relationship of depressive symptoms to symptom reporting, self-care and glucose control in diabetes. Gen Hosp Psychiatry 2003;25:246-52.
68. Gavi S, Hensley J. Diagnosis and management of type 2 diabetes in adults: a review of the ICSI guideline. Geriatrics 2009;64:12.
69. Shoff SM, Klein R, Moss SE, Klein BE, Cruickshanks KJ. Weight change and glycemic control in a population-based sample of adults with older-onset diabetes. J Gerontol A Biol Sci Med Sci 1998;53:M27-32.
70. Ferrand C, Perrin C, Nasarre S. Motives for regular physical activity in women and men: a qualitative study in French adults with type 2 diabetes, belonging to a patients' association. Health Soc Care Community 2008;16:511-20.
71. Donahue KE, Mielenz TJ, Sloane PD, Callahan LF, Devellis RF. Identifying supports and barriers to physical activity in patients at risk for diabetes. Prev Chronic Dis 2006;3:A119-.
72. O'Donovan G, Kearney EM, Nevill AM, Woolf-May K, Bird SR. The effects of 24 weeks of moderate- or high-intensity exercise on insulin resistance. Eur J Appl Physiol 2005;95:522-8.
73. Gucciardi E, Demelo M, Lee RN, Grace SL. Assessment of two culturally competent diabetes education methods: individual versus individual plus group education in Canadian Portuguese adults with type 2 diabetes. Ethn Health 2007;12:163-87.
74. Kang JY, Cho SW, Sung SH, Park YK, Paek YM, Choi TI. Effect of a continuous diabetes lifestyle intervention program on male workers in Korea. Diabetes Res Clin Pract 2010;90:26-33.
75. Uusitupa MI, Stancáková A, Peltonen M, et al. Impact of positive family history and genetic risk variants on the incidence of diabetes: the Finnish Diabetes Prevention Study. Diabetes Care 2011;34:418-23.
76. Uitewaal P, Hoes A, Thomas S. Diabetes education on Turkish immigrant diabetics: predictors of compliance. Patient Educ Couns 2005;57:158-61.

77. Dalewitz J, Khan N, Hershey CO. Barriers to control of blood glucose in diabetes mellitus. Am J Med Qual 2000;15:16-25.
78. Ripsin CM, Kang H, Urban RJ. Management of blood glucose in type 2 diabetes mellitus. Am Fam Physician 2009;79:29-36.
79. St John A, Davis WA, Price CP, Davis TME. The value of self-monitoring of blood glucose: a review of recent evidence. J Diabetes Complications 2010;24:129-41.
80. Hickey MS, Pories WJ, MacDonald KG, J., et al. A new paradigm for type 2 diabetes mellitus: could it be a disease of the foregut? Ann Surg 1998;227:637-43.
81. Chapin RB, Williams DC, Adair RF. Diabetes control improved when inner-city patients received graphic feedback about glycosylated hemoglobin levels. J Gen Intern Med 2003;18:120-4.
82. Huang C, Sousa VD, Chen H, Tu S, Chang C, Pan I. Stressors, depressive symptoms, and learned resourcefulness among Taiwanese adults with diabetes mellitus. Res Theory Nurs Pract 2007;21:83-97.
83. Cox RH, Carpenter JP, Bruce FA, Poole KP, Gaylord CK. Characteristics of low-income African-American and Caucasian adults that are important in self-management of type 2 diabetes. J Community Health 2004;29:155-70.
84. Anonymous Management of hypertension in patients with type 2 diabetes mellitus. Journal of Endocrinology, Metabolism & Diabetes of South Africa 2012;17:S60-1.
85. Chobanian AV, Bakris GL, Black HR, et al. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003;42:1206-52.
86. Anonymous Cardiovascular risk and the management of dyslipidaemia in patients with type 2 diabetes mellitus. Journal of Endocrinology, Metabolism & Diabetes of South Africa 2012;17:S56-8.
87. Reiner Ž, Catapano AL, De Backer G, et al. ESC/EAS Guidelines for the management of dyslipidaemias. European Heart Journal 2011;32:1769-818.
88. Distiller LA. Improved diabetes management in South Africa: the case for a capitation model. Diabetes Voice 2004;49:16-18.
89. Kirkman SM, Williams SR, Caffrey HH, Marraro DM. Impact of a program to improve adherence to diabetes guidelines by primary care physicians. Diabetes care 2002;25:1946-1951.
90. Igbojiaku, O., Harbor, O., Ross,A. Compliance with diabetes guidelines at a regional hospital in KwaZulu-Natal, South Africa. African Journal of Primary Health Care & Family Medicine 2013;5.
91. Joint National Hypertension Guideline Working Group 2006. South African Hypertension Guidelines 2006. SAMJ 2006;96:335-62.
92. Anonymous Screening for and management of chronic kidney disease in adults with diabetes mellitus. Journal of Endocrinology, Metabolism & Diabetes of South Africa 2012;17:S62-5.

93. Anonymous Obesity in type 2 diabetes. Journal of Endocrinology, Metabolism & Diabetes of South Africa 2012;17:S54-5.
94. Anonymous Aspirin therapy in diabetes. Journal of Endocrinology, Metabolism & Diabetes of South Africa 2012;17:S59-.
95. Rabkin R, Ryan MP, Dukworth WC. The renal metabolism of insulin. Diabetologia 1984;27:351-357.
96. Goldfine AB. Statins: Is It Really Time to Reassess Benefits and Risks? N Engl J Med 2012;366:1752-5.
97. Crane PK, Walker R, Hubbard RA, et al. Glucose Levels and Risk of Dementia. N Engl J Med 2013;369:540-8.

6. APPENDIX A – Ethics approval

7. APPENDIX B: Patient consent form

UNIVERSITY OF WITWATERSRAND

CONSENT TO ACT AS A SUBJECT IN RESEARCH

I,_____ being 18 years or older consent to participating in a research project entitled:

PREDICTORS OF GLYCAEMIC CONTROL IN TYPE 2 DIABETES PATIENTS AT HELEN JOSEPH HOSPITAL DIABETIC CLINIC

The procedures/questionnaires have been explained to me and I understand and appreciate their purpose and the extent of my involvement. I have read and understand the attached information leaflet.

I understand that the procedures form part of a research project, and may not provide any direct benefit to me.

I understand that all experimental procedures have been sanctioned by the Committee for Research on Human Subjects, University of the Witwatersrand, Johannesburg.

I understand that my participation is voluntary, and that I am free to withdraw from the project at any time without prejudice.

_____ _____

Participant name and signature Date

_____ _____

Investigator name and signature Date

8. APPENDIX C: Patient information leaflet

INFORMATION DOCUMENT

Study title: Predictors of Glycaemic Control in Type 2 Diabetes Patients at Helen Joseph Hospital Diabetic Clinic

Good day

I am Dr. Daniel Roux and I am doing a research project as part of a degree. Research is just the process to learn the answer to a question. In this study I am trying to find things that determine good control of blood glucose.

I am inviting you to take part in this research study.

Your involvement would consist of a short interview with a few questions, a few measurements (I will take blood pressure, weight, height, measure your waist and neck), record your current medication and also have a look at your latest blood results to see how well your glucose is controlled.

There is absolutely no risk to you by being involved in the study.

You might not see direct benefits of the study but we might learn things from this study that might improve your care later on.

Participation is voluntary. Refusal to participate will involve no penalty or loss of benefits to which you would be entitled. You may discontinue at any time without penalty or loss of benefits. You do not have to answer any of the questions if you do not want to.

Efforts will be made to keep personal information confidential. Absolute confidentiality cannot be guaranteed even though none of your personal identification details will be recorded. Personal information may be disclosed if required by law.
If results are published it may lead to cohort identification.

You are welcome to contact Dr. Daniel Roux at Helen Joseph Hospital if you have any questions or concerns. Phone 011 489 1011 and ask for 50615.

You can also contact the REC administrator and chair – for reporting of complaints or problems at 011 717 1234

9. APPENDIX D: Patient data collection sheet

10. APPENDIX E: Pearson correlation coefficient table

Pearson correlation coefficients										
		HBA_{1C}	Systolic blood pressure	Diastolic blood pressure	Total cholesterol	Triglycerides	HDL-C	LDL-C	Body mass index	Waist circumference
HBA_{1C}	r	1.0	0.02139	0.29981	0.09541	0.13094	-0.05685	0.09329	0.02367	-0.03041
	p		0.8327	**0.0024**	0.3450	0.1941	0.5743	0.3660	0.8152	0.7639
	n	100	100	100	100	100	100	96	100	100
Systolic blood pressure	r	0.02139	1.0	0.55712	0.18590	0.20834	-0.14335	0.12233	0.15645	0.15875
	p	0.8327		**< 0.0001**	0.0640	0.0375	0.1548	0.2351	0.1201	0.1147
	n	100	100	100	100	100	100	96	100	100
Diastolic blood pressure	r	0.29981	0.55712	1.0	0.19193	0.35611	-0.15526	0.09484	0.02298	0.06248
	p	**0.0024**	**< 0.0001**		0.0557	**0.0003**	0.1230	0.3580	0.3580	0.5369
	n	100	100	100	100	100	100	96	100	100
Total cholesterol	r	0.09541	0.18590	0.19193	1.0	0.48292	0.37798	0.86673	0.02097	-0.03117
	p	0.3450	0.0640	0.0557		**< 0.0001**	**0.0001**	**< 0.0001**	0.8359	0.7582
	n	100	100		100	100	100	96	100	100
Triglycerides	r	0.13094	0.20834	0.35611	0.48292	1.0	-0.08284	-0.08284	0.04339	0.10881
	p	0.1941	0.0375	**0.0003**	**< 0.0001**		0.1432	0.4223	0.6682	0.2812
	n	100	100	100	100	100	100	96	100	100
HDL-C	r	-0.05685	-0.14335	-0.15526	0.37798	-0.14746	1.0	0.24368	-0.03362	-0.18846
	p	0.5743	0.1548	0.1230	**0.0001**	0.1432		**0.0167**	0.7398	0.0604
	n	100	100	100	100	100	100	96	100	100
LDL-C	r	0.09329	0.12233	0.09484	0.86673	-0.08284	0.24368	1.0	0.04786	-0.04197
	p	0.3660	0.2351	0.3580	**< 0.0001**	0.4223	**0.0167**		0.6433	0.6847
	n	96	96	96	96	96	96	96	96	96
Body mass index	r	0.02367	0.15645	0.02298	0.02097	0.04339	-0.03362	0.04786	1.0	0.82496
	p	0.8152	0.1201	0.3580	0.8359	0.6682	0.7398	0.6433		**< 0.0001**
	n	100	100	100	100	100	100	96	100	100
Waist circumference	r	-0.03041	0.15875	0.06248	-0.03117	0.10881	-0.18846	-0.04197	0.82496	1.0
	p	0.7639	0.1147	0.5369	0.7582	0.2812	0.0604	0.6847	**< 0.0001**	
	n	100	100	100	100	100	100	96	100	100