Academic Sciences International Journal of Pharmacy and Pharmaceutical Sciences

ISSN- 0975-1491 Vol 4, Suppl 5, 2012

Research Article

CLINICAL CHARACTERISTICS OF DIABETES MELLITUS PATIENTS' SEEKING MEDICAL ADVICE IN OUT-PATIENT DEPARTMENT OF HOSPITAL PENANG, MALAYSIA

SYED WASIF GILLANI[1*], SYED AZHAR SYED SULAIMAN[1], SHAMENI SUNDRAM[2], SITI MAISHARAH SHEIKH GHADZI[3], SABARIAH NOOR HAROUN[3], NUR HAFZAN MD HANAFIAH[3]

[1]School of pharmaceutical sciences, Universiti Sains Malaysia, Pulau Pinang, Malaysia, [2]Doctor, Hospital Pulau Pinang, 10990, Residential Street, Penang, Malaysia, [3]School of Pharmaceutical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan.
Email: wasifgillani@gmail.com

Received: 18 Jun, 2012, Revised and Accepted: 02 Nov, 2012

ABSTRACT

The **aim** of the study was to evaluate the patient clinical characteristics and risk factors for long term complications in the endocrine clinic of Hospital Penang, Malaysia. This study aimed to describe the risk factors and association among sociodemographic; hence **methodology** adopted was Descriptive Prospective Longitudinal study design was chosen. To achieve a power of 0.7 with alpha set at 0.05, 186 subjects were required for the study but researcher increase the sample to 297 in case of drop out. The Research Ethics Committee of hospital and the Malaysian Medical Research and Ethics Committee approved the study. The Statistical package for Social Sciences [SPSS] version 19 ® was used for this analysis. **Findings** A total of 297 [100%] patients were enrolled from OPD diabetic clinic of Hospital Palau Pinang. Among the sample 150 [50.5%] were males and rest 147 [49.5%] females. Mean age comparison among genders showed females have high mean age [59.04 years] as compared to males [58.23 years]. Findings suggested that Malay males has the highest levels of HbA1c and score suggested poor control to DM type II as compared to other ethnics but followed by Indian males and then least mean value found among Chinese males. Whereas among females, Indian showed poor control to DM condition as compared to other ethnics followed by Malays and then least mean score of HbA1c among Chinese females. **Conclusion** Majority of patients already developed long-term complications at the time of diagnosis. Patient with the noncompliance and no adherence issues, probably chronic physiological effect of diabetes mellitus.

Keywords: *Diabetes mellitus, Risk factors, Long term complications, Endocrine clinic, Clinical health.*

INTRODUCTION

The prevalence of diabetes is on the increase and an estimated 239 million people worldwide are expected to have the condition by the year 2020 [1]. Diabetes mellitus [DM] represents a serious health care challenge. It is a heterogeneous disorder characterized by varying degrees of insulin resistance and insulin deficiency, which leads to disturbances in glucose homeostasis. It is commonly associates with prolonged ill health and premature death [2]. The mortality rate in patients with DM may be up to eleven times higher than in persons without the disease [3,4]. DM is the leading cause of blindness, renal failure and foot & leg amputations in adults in developed countries [1].

The World Health Organization [WHO] classification system of DM recognized two major forms of diabetes [5]; Type 1 diabetes mellitus [DM], formerly known as insulin dependent diabetes mellitus [IDDM; patient is dependent on exogenous insulin for survival] and Type 2 DM, formerly known as non-insulin dependent diabetes mellitus [NIDDM; patient is not necessary dependent on exogenous insulin for survival].

Teamwork and collaboration are essential components of successful DM management, both to prevent complications and maintain the patients' health-related quality of life [HRQOL] over a lifetime of coping with the disease [1]. Type 1 DM is characterized by insulin deficiency resulting from immune-mediated pancreatic beta-cell destruction. The most serious acute consequence of this is ketoacidosis. Pancreatic beta-cell destruction eventually results in absolute insulin deficiency [1]. Type 1 DM accounts for approximately ten percent of all DM cases. Type 2 DM is generally characterized by peripheral insulin resistance with relative insulin deficiency to predominant insulin secretory defeat with insulin resistance [1]. Type 2 DM accounts for approximately ninety percent of all DM cases. The major risk factors in the development of type 2 DM are [3]; Family history, Obesity, Race/ethnicity, Increasing age [especially greater than forty five years], Previous identified impaired fasting glucose or impaired glucose tolerance, Hypertension, Hyperlipidemia and History of gestational DM.

There is evidence that good glycaemic control can slow or prevent the development of diabetes complications [6-10]. The Diabetes Control and Complication Trial [DCCT] demonstrated the association between the degree of glycaemic control and the development of microvascular complications in type 1 DM patients [11-12]. The DCCT determined that there was an approximately 50% reductions in microvascular complications in the intensive treatment group and a non-significant tendency to fewer major cardiovascular events. Intensive control was accompanied by a significantly between the groups. The DCCT investigators did advice caution in extending the findings to patients with type 2 DM without careful regard for age and coexisting diseases.

The United Kingdom Prospective Diabetes Study [UKPDS] was the largest scale long-term intervention study in newly diagnosed type 2 DM patients and involved over 5000 patients. The UKPDS used an intensive blood glucose control policy, which achieved a medium HbA1c of 7% compared with 7.9% in those randomized to conventional treatment over a median 10 years follow-up [9]. The UKPDS confirmed the benefit of intense glycaemic control on microvascular disease in type 2 DM patients [4, 8-10, 13-20]. The aim of the study was to evaluate the patient clinical characteristics in the endocrine clinic of Hospital Penang, Malaysia.

METHODS

Method design

This study aimed to describe the risk factors and association among sociodemographic, hence Descriptive Prospective Longitudinal study design was chosen.

Setting

As 70% of people with diabetes in Malaysia receive treatment in the government healthcare system, [21] data was collected from government healthcare settings. The general hospital is the main government hospital in the Penang state and is situated within the city area offering tertiary care. Subjects were not recruited from private clinics and hospitals due to problems with accessibility and differences in socio-economic status which could bias the outcomes.

Sample Size

The required sample size was calculated with power analysis using the procedure provided by the Polit and Hungler [22]. The power was

set at 0.80 with alpha being set at 0.05. Since the value of the effect size [Gamma], was unavailable from previous similar studies and the pilot study sample size was small [19 subjects], the investigator chose to use the conversion based on the effect size convention table in Polit and Hungler [2004, p495]. [22] Polit and Hungler [2004] advise to use medium effect size ranging from 0.2-0.3 for nursing studies [22]. This provided a range of sample size from 88-197 subjects. For logistical reasons the study had to be a manageable size within the period of study, so the investigator chose the sample size using the medium effect size of Gamma y = 0.25. To achieve a power of 0.7 with alpha set at 0.05, 186 subjects were required for the study but researcher increase the sample to 297 in case of drop out.

Inclusion Criteria

Subjects who met the following criteria were recruited. They were:

- non-pregnant adults with either Type1 or Type 2 diabetes regardless of gender and ethnicity
- 18 years and above [legal age for consent]
- Diagnosed with diabetes with year of more
- Speaking and understanding English, Bahasa Malaysia, Mandarin, Chinese dialects [Cantonese, Hokkien, Teow-chew] because these were the languages used during the interview.
- having poor diabetes control during the last one year*

*Even though glycated haemoglobin [HbA1c] is the gold standard for glycaemic assessment, it was not consistently measured for all diabetic patients in the healthcare system where the study was done. Therefore for the purpose of this study, poor diabetes control was defined as the mean of minimum of three fasting blood glucose [FBG] readings of more than 7 mmol/L in the last year. Prior studies have shown that FBG of more than 7 mmol/L is associated with increased micro-and macro-vascular complications [23-26].

Exclusion criteria

The following subjects were excluded. They:

- Were adults 18 years of age and more with either Type 1 of Type 2 diabetes unable to answer the questionnaires independently, such as having unstable medical condition, mental illness, and senility or hearing impairment. This was to avoid assistance from family members to cares that could introduce bias in the data collection.
- Had poor vision and unable to assess visually the portion sizes of their carbohydrate food intake during dietary assessment
- Were women who were pregnant or had gestational diabetes due to different criteria on standard of control
- Had record of random blood glucose only because the definition of poor control was based on fasting blood glucose readings.

Research Tool

Self-developed data collection form was used to collect the patient information.

Ethical Issues

The Research Ethics Committee of hospital and the Malaysian Medical Research and Ethics Committee approved the study.

Written consents which included information to access the subjects' medical records were taken from all participants before the interviews. For those who were illiterate and not able to give their signature, thumbprints were used instead.

Data Collection Procedure

Identification of Subjects

This was done initially by identifying all diabetic subjects. In the out-patient department of the hospital, the investigator worked closely with the nursing staff to identify patients with blood glucose tests done prior to doctors' consultation. They were familiar with their patients with poor glycaemic control or the nurse in-charge identified them via the patients' blood glucose results.

Places of Data Collection

Data collection was done in out-patient departments at the doctors' consultation rooms.

Statistical Analysis

The analysis was done in both descriptive and inferential statistics to make information in presentable form. Data is presented in both graphical and tabular forms. The Statistical package for Social Sciences [SPSS] version 19 ® was used for this analysis. The level of significance was set at 0.05 for all analysis.

RESULTS

A total of 297 [100%] patients were enrolled from OPD diabetic clinic of Hospital Palau Pinang. Among the sample 150 [50.5%] were males and rest 147 [49.5%] females. Mean age distribution among gender is presented in table 1.

Table 1: Frequency among gender of study population

Gender	F [%]	Age Mean [± S.D]
Male	150 [50.5]	58.23 [10.771]
Female	147 [49.5]	59.04 [10.414]
Total	297 [100.0]	58.64 [10.581]

Ethnic distribution among males showed predominance of Chinese with 77 [51.3%] followed by Malays 40 [26.7%] and rest 33 [22%] were Indians. While among females almost same pattern was found. The mean ± S.D age differences were found variable among the three ethnics. Table 2 showed the distribution pattern of mean ± S.D of age among genders and also among the study population.

Table 2: Mean age gender distribution among ethnic

Gender	Ethnic	Mean	N [%]	Std. Deviation
Male	Malay	53.20	40[26.7]	12.831
	Chinese	62.10	77[51.3]	8.612
	Indian	55.03	33[22]	9.600
	Total	58.23	150 [50.5]	10.771
Female	Malay	54.03	36[24.49]	8.013
	Chinese	63.43	81[55.10]	11.090
	Indian	58.30	30[20.41]	9.063
	Total	59.04	147[49.5]	10.414
Total	Malay	53.61	76[25.59]	10.627
	Chinese	62.75	158[53.20]	9.959
	Indian	56.53	63[21.21]	9.422
	Total	58.64	297 [100.0]	10.581

Mean age comparison among genders showed females have high mean age [59.04 years] as compared to males [58.23 years]. While comparing mean age difference among ethnics revealed Chinese mean age is 62.75 years followed by Indians with 56.53 years and least age to disease response among Malays 53.61 years. Mean weight of the study population was 66.29kg but upon analysis among genders it is found that males mean ± S.D [70.34 ± 15.185] is extensively higher as compared to females [62.37 ± 13.382].

Further analysis by cross tabulation showed Malay males mean weight at the time of diagnosis significantly higher [p<0.001, one way ANOVA] as compared to other ethnics, same results found among Malay females [p<0.001, one way ANOVA]. Table 3 showed cross-tabulation of mean weight distribution among gender and ethnics at the time of diagnosis. At the time of diagnosis body mass index [BMI] has been collected and results showed the mean BMI of the study population was 25.39 [297, 100%], among them females have higher BMI 25.79 as compared to males 24.97 at the time of diagnosis.

Table 3: Mean weight in kg at diagnosis gender distribution among ethnic

Gender	Ethnic	Mean	N [%]	Std. Deviation
Male	Malay	79.17	40[26.67]	18.936
	Chinese	66.64	77[51.33]	13.003
	Indian	69.19	33[22]	11.314
	Total	70.34	150 [50.5]	15.185
Female	Malay	69.38	36[24.49]	14.896
	Chinese	60.53	81[55.10]	12.766
	Indian	59.95	30[20.41]	11.069
	Total	62.37	147[49.5]	13.382
Total	Malay	74.74	76[25.59]	17.761
	Chinese	63.31	158[53.20]	13.184
	Indian	64.96	63[21.21]	12.019
	Total	66.29	297 [100.0]	14.815

HbA1c is a lab test that shows the average amount of sugar in the blood over past 3 months. It shows how well patient is controlling its diabetes condition. Our findings from the HbA1c showed that females controlled DM type II well as compared to males but overall mean ± S.D of HbA1c among study population reflects poor compliance.

Findings suggested that Malay males has the highest levels of HbA1c and score suggested poor control to DM type II as compared to other ethnics but followed by Indian males and then least mean value found among Chinese males. Whereas among females, Indian showed poor control to DM condition as compared to other ethnics followed by Malays and then least mean score of HbA1c among Chinese females.

Study findings showed that FBS mean value is quiet high at the time of diagnosis, such results referred to late diagnosis of DM type II and would be consider as chronic cases. Overall females of the study population have significantly ($p<0.01$, Student T-Test) higher mean FBS value as compared to males. Table 4 provides the information of mean FBS distribution among gender of study population at the time of diagnosis. On further analysis findings suggested that both Malay males and females have significantly ($p<0.01$, one way ANOVA) high mean values of FBS followed by Indians and then least mean score value with Chinese. Study population was diagnosed with respect to different classes, findings suggested that majority of the patients seek medical attention with complications (HPT, others). On analysis it was found that majority of both males and females had clinical complications at the time of diagnosis only 92 (31.0%) patients seek medical advice with diabetes alone (Table 4, 5).

Table 4: Type of diabetic mellitus among gender in study population at time of diagnosis

		Gender		Total N (%)
		Male N (%)	Female N (%)	
Diagnosis Class	IDDM	1 (33.3)	2 (66.7)	3 (1.0)
	Diabetes alone	49 (55.1)	40 (44.9)	89 (30.0)
	Diabetes with HPT	49 (48.5)	52 (51.5)	101 (34.0
	Diabetes with Other complications	51 (49.0)	53 (51.0)	104 (35.0)
Total		**150 (50.50)**	**147 (49.5)**	**297 (100.0)**

* Insulin dependent Diabetes mellitus (IDDM)

Table 5: Type of diabetic mellitus among gender and ethnic at the time of diagnosis

Gender			Race				P value
			Malay N (%)	Chinese N (%)	Indian N (%)	Total N (%)	
Female	Diagnosis Class	Diabetes with Other complications	12 (23.0)	30 (57.7)	10 (19.3)	52 (100.0)	
		Diabetes with HPT	12 (23.0)	33 (63.5)	7 (13.5)	52 (100.0)	
		Diabetes alone	11 (26.7)	18 (44.0)	12 (29.3)	41 (100.0)	.619
		IDDM	1 (50.0)	-	1 (50.0)	2 (100.0)	
	Total		**36 (24.5)**	**81 (55.10)**	**30 (20.40)**	**147 (100.0)**	
Male	Diagnosis Class	Diabetes with Other complications	8 (15.7)	33 (64.7)	10 (19.6)	51 (100.0)	
		Diabetes with HPT	14 (28.6)	27 (53.1)	8 (18.3)	49 (100.0)	**.048**
		Diabetes alone	18 (36.7)	17 (34.7)	14 (28.6)	49 (100.0)	
		IDDM	-	-	1 (100)	1 (100.0)	
	Total		**40 (26.7)**	**77 (51.3)**	**33 (22.0)**	**150 (100.0)**	

*Chi-Square (Fisher Exact Test)

In the cross comparison of dependent variables with the diabetes class showed significant results except FBS, findings suggested the FBS is the identical and individual functional parameter among diabetes patients. Analysis also showed that FBS has no effect on the class of diagnosis, patient social habit might influence the FBS. Table 6 showed the detailed information of cross-comparison of diagnosis class with different dependent variables; association was assessed by using One way ANOVA.

Table 6: Diagnosis associated variables with relative significant value (using One way ANOVA)

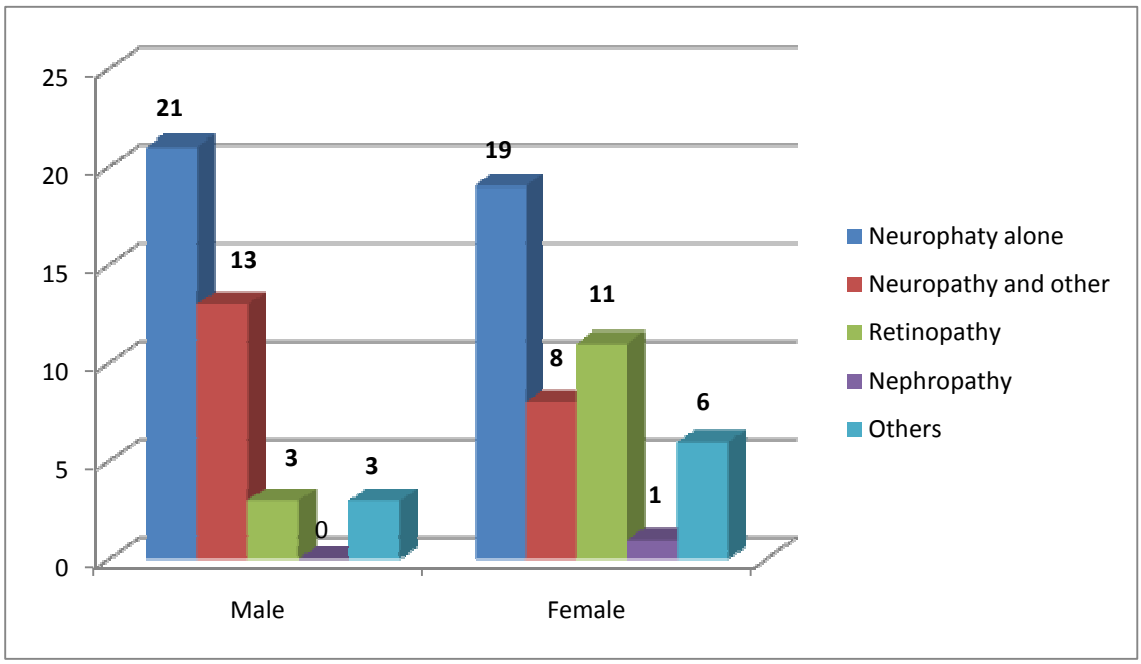
Diagnosis	Age Mean ± S.D	P value	Weight in kg Mean ± S.D	P value	HbA1C Mean ± S.D	P value	BMI Mean ± S.D	P value	FBS Mean ± S.D	P value
IDDM	58.00±6.083	.000	64.00±11.533	.007	10.16±4.041	.029	24.16±3.013	.009	9.40±1.49	.268
Diabetes	53.93±11.484		69.56±15.889		8.719±3.021		26.05±4.580		9.47±3.54	
Diabetes with HPT	60.17±10.849		68.63±16.531		7.927±2.305		26.42±5.259		7.95±3.44	
Diabetes with Other complications	61.06±8.217		62.14±11.319		7.567±2.020		24.10±3.987		9.56±9.14	

It has been seen that majority 246 (82.83%) of the patients received oral therapies for the treatment of DM type II. Prescribing pattern among gender showed similar results. On further assessment with ethnic distribution among gender and medication consider, showed significant association with female population among Chinese.

A total of 85(28.7%) patients seek medical advice with long-term complications. Among them 61(71.8%) patients had neuropathy and related complications. Table 7 shows the descriptive information related to clinical complications at the time of diagnosis and graph 1 show the gender differences related to clinical complications at the time of diagnosis.

Table 7: Estimated incidence of clinical complication among study population at the time of diagnosis (N = 85)

Complications	N (%)
Neuropathy	40 (47.1)
Neuropathy and others complications	21 (24.7)
Retinopathy	14 (16.5)
Nephropathy	1 (1.2)
Others	9 (10.5)
Total	**85 (100.0)**



Graph 1: Descriptive distribution of clinical complications in gender at the time of diagnosis

In term of incidence of long-term hyperglycemic complications at the time of diagnosis, there is no significant difference among genders but with in the third layer comparison between complications among ethnicity showed significant (p< 0.024) differences as Chinese have high incidence of complications both in male and female gender as compared to other ethnics (Table 5).

In the current stage of analysis it is important to know the selection of antihyperglycemic medication, to be prescribed in the known preliminary clinical complications among study population. The selection and compliance of medication would reflect the chronicity of the disease and related body response to the current stage of disease. Often malclinical practices also performed in the selectivity of medication. Table 8 presented a detail list of medications used among study population with used combinations. The data presented in the interpretation of maintenance dose with controlled FBS or HbA1c value.

Table 8: Cross-comparison of clinical complications among gender and ethnic distribution of study population

Gender			Ethnic			Total N (%)
			Malay N (%)	Chinese N (%)	Indian N (%)	
Male	**Complications**	Neuropathy	4 (19.0)	11 (52.4)	6 (28.6)	21 (100.0)
		Neuropathy and other	2 (15.4)	5 (38.5)	6 (46.1)	13 (100.0)
		Retinopathy	-	2 (66.7)	1 (33.3)	3 (100.0)
		Others	1 (33.3)	2 (66.7)	-	3 (100.0)
	Total		**7 (17.5)**	**20 (50.0)**	**13 (32.5)**	**40 (100.0)**
Female	**Complications**	Neuropathy	6 (31.6)	9 (47.4)	4 (21.2)	19 (100.0)
		Neuropathy and other	2 (25.0)	5 (62.5)	1 (12.5)	8 (100.0)
		Retinopathy	-	10 (90.9)	1 (9.1)	11 (100.0)
		Nephropathy	-	1 (100.0)	-	1 (100.0)
		others	1 (16.7)	4 (66.7)	1 (16.6)	6 (100.0)
	Total		**9 (20.0)**	**29 (64.4)**	**7 (15.6)**	**45 (100.0)**

p value : Fisher exact test (Male 0.699/ Female 0.242)

Majority (54.0%) of study population showed non-compliance to medication given. Findings of the analysis suggested that patient mean weight and BMI at the time of diagnosis are the significant variables influencing the compliance status of the patient. Higher the value of associated variable more prone towards non-compliance. Comparison was made between compliance to medication and demographic & preliminary clinical characteristics (Table 9). Findings showed significant association of compliance to medication with the class of diagnosis and preferred medication at the time of diagnosis.

Table 9: Prescribed antihyperglycemic medication and combination in study population

Prescribed medication	N (%)
Metformin[a]	105 (35.4)
Metformin with diamicron[b]	57 (19.1)
Metformin with glicazide[c]	49 (16.5)
Metformin with daonil[d]	18 (16.1)
Glicazide[e]	33 (11.1)
Others combination[f]	30 (10.1)
Diet control	5 (1.7)
Total	**297 (100.0)**

[a]250 mg BD, 500 mg BD, 750 mg BD, 1g BD

[b] 80/500mg BD, 160/1g BD, 120/1g BD, 120/500 BD, 80/750 BD, 40/1g BD

[c]80 mg/500mg BD, 80mg/250mg BD, 120/750 BD, 100tds/500bd, 40/500 BD, 80/1g BD, 120/250 BD, 40/250 BD, 160/500 BD, 20/500 BD, 40/750 BD

[d] 5mg/500mg BD, 10/750 BD, 2.5/500 BD, 10/1g BD, 10/500 BD, 7.5/1g BD, 10/250 BD, 5/250mg BD

[e] 160 mg BD, 40 BD, 120BD, 80 BD

[f] - s/c Insulin 35 iU ON with Diamicron 160 BD and Metformin 1g BD

- acarbose 50mg TDS

- daonil (2.5 mg OM, 5 mg BD, 7.5mg BD)

- s/c insulatard 28/16 iU with actaprid 12/6 iU

- s/c insulatard 20/14 iU with actaprid 10/6 iU

- s/c insulatard 28/28 iU with actaprid 16/10 iU with metformin 1g

- s/c insulatard 14 iU ON with 1 g metformin BD

- diamicron 80mg OM 40mg ON

- Glibenclimide 7.5 mg OM 5 mg ON

- Glocovance

-repaglinide 1mg TDS with metformin 1g BD

In comparison between adherence with demographic and preliminary clinical characteristics. Findings showed significant association of compliance to medication with the class of diagnosis, preferred medication at the time of diagnosis and also ethnicity. Chinese are more adherent to therapy followed by Indians and then Malays. Further analysis also suggested that patient mean age and HbA1c during treatment are the significant variables influencing the compliance status of the patient. Higher the value of mean age more prone to adherent as compared to low mean age. (Table 10)

Table 10: Characteristic determination of compliance in study population

Characteristic	Compliance		Total N(%)	P-value
	Yes N(%)	No N(%)		
Gender				
Male	67 (44.7)	83 (55.3)	150 (100.0)	.117
Female	81 (55.1)	66 (44.9)	147 (100.0)	
Ethnic				
Malay	34 (44.7)	42 (55.3)	76 (100.0)	.159*
Chinese	85 (54.5)	71 (45.5)	156 (100.0)	
Indian	27 (41.5)	38 (58.5)	65 (100.0)	
Diagnosis				
IDDM	1 (33.3)	2 (66.7)	3 (100.0)	.010
Diabetes	38 (42.7)	52 (57.3)	89 (100.0)	
Diabetes and HPT	47 (46.5)	54 (53.5)	101 (100.0)	
Diabetes and other complication	61 (58.6)	43 (41.4)	104 (100.0)	
Medication consideration				
Insulin	3 (50.0)	3 (50.0)	6 (100.0)	.040*
Insulin and oral	5 (20.8)	19 (79.2)	24 (100.0)	
BIDS	8 (44.4)	10 (55.6)	18 (100.0)	
Oral	128 (52.0)	118 (48.0)	246 (100.0)	
Diet and exercise	2 (66.7)	1 (33.3)	3 (100.0)	

Chi-square (*Fisher exact test)

DISCUSSION

Diabetic neuropathy is a common complication of diabetes. It usually progresses gradually and involves small and large sensory fibers. The symptoms, such as loss of ability to sense pain, loss of temperature sensation, and developing neuropathic pain, follow a "glove and stocking" distribution, beginning in the lower limbs, first affecting the toes, and then progressing upward. [27]. The primary cause of diabetic neuropathy is thought to be hyperglycemias [28].

Diabetic neuropathy represents a major health problem worldwide. An Australian population based survey of 2436 patients with known or newly diagnosed diabetes showed that 13.1% of them had peripheral neuropathy [29]. Another multicentre study in the United Kingdom showed that 22-32% of 6363 diabetic patients had peripheral neuropathy [30]. Similar results have been reported by an Italian multicentre study, which showed that 32.3% of 8757 diabetic patients had neuropathy [31]. Symptoms of neuropathic pain are commonly reported in patients with diabetic neuropathy. Partanen and colleagues found that among 132 patients, 7-13% had pain and paraesthesias when they were diagnosed as having type 2 diabetes mellitus [32]. The prevalence's of pain and of paraesthesia were 20% and 33% 10 years after diagnosis [32]. Sorensen and colleagues identified neuropathic pain in 11.7% of those who had insensate neuropathy and in 2.3% of those with sensate neuropathy among 2610 patients with type 2 diabetes [33]. Tight glycaemic control has been shown to be effective in slowing the progression of diabetic neuropathy [34-37]. The diabetes control and complications trial in 1441 patients with type 1 diabetes showed that tight glycaemic control can delay the onset and slow the progression of neuropathy, as measured by clinical examination, autonomic testing, and nerve conduction studies [36-37]. Apart from glycaemic control, antidepressants and anticonvulsants are commonly

used to reduce the intensity of pain in patients with painful diabetic neuropathy. Painful symptoms reported by patients with diabetic neuropathy have been frequently documented. Neuropathic pain symptoms are reported in 3-20% of patients with diabetic neuropathy [32-33,38-39]. Pain paroxysms, deep aching pain, and hot or burning pain have often been described 39-40. In the clinical setting, management focuses on two aspects: disease modifying treatment such as glycaemic control and the use of various kinds of analgesics to reduce the intensity of the pain. Although pain intensity may not be sufficient to reflect the outcome of treatment, it is a common outcome measure in clinical research. Few studies reported treatment efficacy for different qualities of pain such as allodynia and burning pain [30,37,42]. The efficacy of drug treatment may be underestimated, especially for particular painful symptoms. In our study we come to discover that majority of patients 61 (71.8%) presented with neuropathy and related complications, it would be a predictor for long term complication among type II diabetes mellitus.

The epidemiology of DN has been best studied in patients with type 1 diabetes, because the time of clinical onset is usually known. Approximately 25% to 45% of these patients develop clinically evident disease during their lifetime [43-45]. The peak time to development of nephropathy in type 1 diabetes is between 10 and 15 years after the onset of disease. Importantly, patients who do not develop proteinuria after 20 to 25 years of diabetes have a very low subsequent risk of developing overt renal disease of only about 1% per year [43]. In patients with type 2 diabetes, the prevalence of progressive renal disease has previously been reported to be lower. Nephropathy develops in up to 50% of type 2 diabetic Pima Indians 20 years after diagnosis of type 2 diabetes, however, and 15% have progressed to ESRD by this time [46-47]. Importantly, proteinuria is a risk factor for cardiovascular disease and it is possible that previous studies underestimate the prevalence of DN in type 2 diabetes because many patients died of cardiovascular disease before developing ESRD.

Recent data suggest that the risk of nephropathy is equivalent in the two types of diabetes. Evidence in support of this hypothesis in one report were the observations that the time to proteinuria from the onset of diabetes and the time to ESRD from the onset of proteinuria were similar in type 1 and type 2 disease [48].

Diabetic retinopathy is more prevalent among patients with type 1 diabetes than type 2. Within 5 and 10 years of diagnosis, about 58% and 80%, respectively, have retinopathy. After 15 to 20 years of disease, more than 90% have some kind of retinopathy and approximately 60% have proliferative retinopathy. After greater than or equal to 20 years 99% have retinopathy and 53% have proliferative retinopathy. In comparison, more than 25% of patients with type 2 diabetes have retinopathy within 2 years of diagnosis. Sixty percent have some retinopathy and 5% have proliferative retinopathy greater than or equal to 20 years after diagnosis, far less than type 1 diabetes [49].

Diabetic nephropathy (DN) and diabetic retinopathy (DR) are arguably the two most dreaded complications of diabetes. Together they contribute to serious morbidity and mortality. As they progress to end-stage renal disease (ESRD) and blindness, they impose enormous medical, economic, and social costs on both the patient and the health care system. Because nephropathy and retinopathy are frequently linked in patients, this article reviews their common and individual aspects of pathophysiology, clinical features, and management. Diabetic nephropathy is a clinical syndrome characterized by persistent albuminuria, arterial blood pressure elevation, a relentless decline in glomerular filtration rate (GFR), and a high risk of cardiovascular morbidity and mortality. This major life-threatening complication develops in approximately 20% to 40% of type 1 and less than 20% of type 2 diabetic patients [50]. DN is the leading known cause of ESRD in the United States, accounting for an estimated 28,000 new cases of ESRD per year [50]. Retinopathy is a serious microvascular complication of diabetes mellitus and the leading cause of blindness in adults less than 65 years of age. It is estimated that about 5.5 million adult patients with diabetes have DR. About 50,000 new cases of blindness occur per year, out of which 50% are caused by diabetes and most caused by DR [51]. Nevertheless, the impact of these complications remains significant and clinicians should remain vigilant. Regular screening as recommended by guidelines and prompt institution of treatment lead to further reductions in morbidity and mortality.

Our study reported the direct influence of BMI on the FBS, also sociodemographic differences in the management treatment outcomes. A positive correlation between BMI and blood sugar was also reported by other studies [52-53]. Ethnicity affects the association between obesity and diabetes and that probably explains the different levels of association between obesity and blood glucose levels which are observed in various studies [54]. The mean BMI of different age groups showed an increasing trend over the decades and an increase in mean BMI was found to be more marked from the 3rd to the 4th decade. The prevalence of obesity, as measured by BMI, is high in many countries all over the world and is rising. It is mainly attributed to the changing lifestyles and dietary habits [55-56]. Mean FBS increased with increasing age and with increasing BMI. Significant increase in mean FBS was observed during the 4th decade of life.

The mechanism by which obesity induces insulin resistance is poorly understood, but a number of mechanisms have been suspected to be involved. Obesity causes peripheral resistance to insulin-mediated glucose uptake and may also decrease the sensitivity of the beta-cells to glucose [57]. These changes are largely reversed by weight loss, leading to a fall in blood glucose concentrations towards normal levels. Weight gain precedes the onset of diabetes; conversely, weight loss is associated with a decreased risk of type 2 diabetes [58-59]. The administration of resistin, an adipocyte derived hormone, decreases while the neutralization of resistin increases insulinmediated glucose uptake by the adipocytes. Thus, resistin may be a hormone that links obesity to diabetes [60]. Leptin is produced by adipocytes and is secreted in proportion to the adipocyte mass. It signals the hypothalamus about the quantity of stored fat. Studies in humans and animals have shown that leptin is associated with obesity and insulin resistance [61]. The deficiency of adiponectin, an adipocyte-derived hormone, plays a role in the development of insulin resistance and subsequently, type 2 diabetes [62]. Retinol-binding protein 4, free fatty acids, tumour necrosis factor-alpha, plasminogen activator inhibitor 1, interleukin-1 beta, uncoupling protein 2 and obestatin are also implicated in the adipose tissue induced pathogenesis of type 2 diabetes [63]. BMI is a good measure of adiposity; however, the relationship between actual body fat and BMI differs between ethnic groups, and as a consequence, the cut off points for the overweight status and obesity based on BMI, will have to be ethnicity specific [64].

Our study also mentioned the noncompliance and non-adherence issues among the study population. A systematic review to

summarize the factors associated with poor control of diabetes. Life style modification is one of the major determinants of diabetes control [65]. In review elderly patients having (> 60 years), males and having normal BMI patients had better control on diabetes. Probably, younger diabetics did not care about the disease control. Usually, the females take the disease only as a second priority as compared to males [66].

Presence of diseases like coronary heart disease, neuropathy, retinopathy, renal failure and neurological disorders was associated with poor control of diabetes [67]. This shows the importance of diabetes control to prevent complications. Foot problems and fatty liver were not related to poor control of diabetes [19]. Probably there could be other factors that are responsible for poor control of diabetes. With the use of insulin, the control of diabetes improves [67-68]. Metformin reduces insulin resistance, thereby improving diabetes control [68]. Surprisingly, poorly controlled patients were more adhered to diet, exercise, medication and regular glucose monitoring [69]. One of the reasons could be that once these patients notice that their diabetes is poorly controlled, they are more likely to get adhered to the good behavior.

CONCLUSION

Majority of patients already developed long-term complications at the time of diagnosis. It has been found that increased rate of risk factors have been found among the study population and non-significant to sociodemographic differences. Patient with the noncompliance and no adherence issues, probably chronic physiological effect of diabetes mellitus.

REFERENCES

1. Patel A. Diabetes in focus. London: Pharmaceutical Press; 1999.
2. Douglas E, Bemiie M, MeAnaw J, Hudson S. Diabetes mellitus. *Pharm J* 1998;261:810-7.
3. Florence JA, Yeager BF. Treatment of type 2 diabetes mellitus. *Am Fam Phys* 1999;59:2835-44.
4. UK Prospective Diabetes Study Group. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 38. *BMJ* 1998;317:703-13.
5. King H, Rewers M, WHO Ad Hoc Diabetes Reporting Group. Global estimates for prevalence of diabetes mellitus and impaired glucose tolerance in adults. *Diabetes Care 1993;16:157-77.*
6. Keen H. Impact of new criteria for diabetes on pattern of disease. *Lancer* 1998;352:1000-l.
7. Miller M. Type II diabetes: A treatment approach for the older patient. *Geriatrics* 1996;5 1:43-9.
8. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with suiphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet 1998;352:837-53.*
9. Stratton lM, Alder Al, Neil HAW, Matthews DR, Manley SE, Cull CA, et al. Association of glycaeinia with macrovascular and microvascular complications of type 2 diabetes (UKPDS *35):* prospective observational study. BMJ2000;321:405-12.
10. Turner R, Millns H, Neil H, Stratton I, Manley S, Matthews D, et al. Risk factors for coronary artery disease in non-insulin dependent diabetes mellitus: United Kingdom prospective diabetes study (UKPDS:23). *BMJ* 1998;316:823-8.
11. Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *New Engl JMed* 1993:329:977-86.
12. Diabetes Control and Complications Trial Research Group. Incidence of intensive diabetes treatment in quality of life outcomes in the diabetes control and complications trial. *Diabetes Care* 1996;19:l95-203.
13. UK Prospective Diabetes Study (UKPDS) Group. Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). *The Lancet* 1998;352:854-65.
14. Davis T, Cull C, Holman R, The U.K. Prospective Diabetes Study (UKPDS) Group. Relationship between ethnicity and glycaemic control, lipid profiles and blood pressure during the first nine years of type 2 diabetes: U.K. Prospective Diabetes Study (UKPDS *55). Diabetes Care* 2001;24:1 167-74.
15. Nathan DM. Some answers, more controversy, from UKPDS. *Lancet* 1998;352:832-3.
16. UK Prospective Diabetes Study Group. Efficacy of atenolol and captopril in reducing risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 39. *BMJ* 1998;317:713-20.
17. UK Prospective Diabetes Study Group. Quality of life in type 2 diabetic patients is affected by complications but not by intensive policies to improve blood glucose or blood pressure control (UKPDS 37). *Diabetes Care 1999;22:1125-36.*
18. UK Prospective Diabetes Study Group. UKPDS 17. A nine-year update of a randomized, controlled trial on the effect of improved metabolic control on complications in non-insulin-dependent diabetes mellitus. *Aim Intern Med* 1996;124:136-45.
19. Turner R, Cull C, Frighi V, Holman R. Glycaemic control with diet, sulfonylurea, metformin, or insulin in patients with type 2 diabetes mellitus: progressive requirement for multiple therapies (UKPDS 49). *JAMA* 1999;281 :2005-12.
20. Adler A, Stratton I, Neil H, Yudkin J, Matthew D, Cull C. Association of systolic blood pressure with macrovascular and microvascular complications of type 2 diabetes (UKPDS 36): prospective observational study. *BMJ2000;321:412-9.*
21. Merican MI, Rohaizat Y, Haniza S. Developing the Malaysian Health System to meet the challenges of the future. Medical Journal of Malaysia 2003;59(1):84-93.
22. Polit DF, Hungler BP. Nursing research-principle and methods Philadelphia: Lippincott Williams & Wilkins; 2004.
23. Cockram C. The epidemiology of diabetes mellitus in the Asia-Pacific region. Hong Kong Medical journal 2000; 6(1):43-52.
24. Arcavi L, Behar S, Caspi A, Reshef N, Boyko V, Knobler H. High fasting glucose levels as a predictor of worse outcome in patients with coronary artery disease: results from the Benzfibrate Infarction prevention (BIP) study. American Heart Journal 2004; 147:239-245.
25. Danaei G. Murray C, Ezzati M, Lawes C, Vander Hoom S. Global and regional mortality from ischemic heart disease and stroke attributable to higher than optimum blood glucose concentration: comparative risk assessment. Lancet 2006;368:1651-1659.
26. Weinger K, Butler HA, Welch GW, LA Greca AM. Measuring diabetes self-care; a psychometric analysis of the self-care inventory-revised with adults. Diabetes Care 2005;28(6):1346-1352.
27. Greene DA, Stevens MJ, Feldman EL. Diabetic neuropathy: scope of the syndrome. Am J Med 1999;107:2-8S.
28. Klein C, Polydefkis M, Chandhry V. Peripheral neuropathy treatment trials. In: Biller J, Bogousslavaky J, eds. Blue books of practical neurology: clinical trials in neurologic practice. USA: Butterworth- Heinemann, 2001:261-91.
29. Tapp RJ, Shaw JE, de Courtenm MP, Dunstan DW, Welborn TA, Zimmet PZ, et al. Foot complications in type 2 diabetes: an Australian population-based study. Diabet Med 2003;20:105-13.
30. Young MJ, Boulton AJM, Williams DRR, Mcleod AF, Sonksen PH. A multi-center study of the prevalence of diabetic neuropathy in patients attending UK diabetic clinics. Diabetologia 1993;36:150-4.
31. Fedele D, Comi G, Coscelli C, Cucinotta D, Feldman EL, ChirlandaG, et al. Amulticenter study on the prevalence of diabetic neuropathy in Italy. Diabetes Care 1997;20:836-43.
32. Partanen J, Niskanen L, Lehtinen J, Mervaala E, Siitonen O, Uusitupa M. Natural history of peripheral neuropathy in patientswith non-insulin-dependent diabetes mellitus.N Engl J Med 1995;333:89-94.
33. Sorensen L, Molyneaux L, Yue DK. Insensate versus painful diabetic neuropathy: the effects of height, gender, ethnicity and glycemic control. Diabetes Res Clin Pract 2002;57:45-51.
34. OhkuboY, KishikawaH,Araki E,MiyataT, IsamiS,Motoyoshi S, et al. Intensive insulin therapy prevents the progression of

diabetic microvascular complications in Japanese patients with non-insulindependent diabetes mellitus: a randomized prospective 6-year. Diabetes Res Clin Pract 1995;28:103-17.
35. Reichard P, Nilsson BY, RosenqvistU. The effect of long-term intensified insulin treatment on the development of microvascular complications of diabetes mellitus.N Engl J Med 1993;329:304-9.
36. Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus.N Engl J Med 1993;329:977-86.
37. Diabetes Control and Complications Trial Research Group. The effect of intensive diabetes treatment on the development and progression of neuropathy. Ann InternMed 1995;122:561-8.
38. Boulton AM, Knight G, Drury J, Ward JD. The prevalence of symptomatic diabetic neuropathy in an insulin treated population. Diabetes Care 1985;8:125-8.
39. Daousi C,MacFarlane IA, Woodward A, Nurmikko TJ, Bundred PE, Benbow SJ. Chronic painful peripheral neuropathy in an urban community: a controlled comparison of people with and without diabetes. Diabet Med 2004;21:976-82.
40. Galer BS, Gianas A, Jensen MP. Painful diabetic polyneuropathy: epidemiology, pain description, and quality of life. Diabetes Res Clin Pract 2000;47:123-8.
41. Otto M, Bak S, Bach FW, Jensen TS, Sindrup SH. Pain phenomena and possible mechanism in patients with painful polyneuropathy. Pain 2003;101:187-92.
42. Ertas M, Sagduyu A, Arac N, Uludag B, Ertekin C. Use of levodopa to relieve pain from painful symmetrical diabetic polyneuropathy. Pain 1998;75:257-9.
43. Ismail N, Becker B, Strzelczyk P, Ritz E. Renal disease and hypertension in non–insulindependent diabetes mellitus. Kidney Int 1999;55:1–28.
44. Orchard TJ, Dorman JS, Maser RE, Becker DJ, Drash AL, Ellis D, et al. Prevalence of complications in IDDM by sex and duration. Pittsburgh Epidemiology of Diabetes Complications Study II. Diabetes 1990;39:1116–24.
45. Parving HH, Hommel E, Mathiesen E, Skott P, Edsberg B, Bahnsen M, et al. Prevalence of microalbuminuria, arterial hypertension, retinopathy and neuropathy in patients with insulin dependent diabetes. BMJ 1988;296:156–60.
46. Nelson RG, Knowler WC, Pettitt DJ, Saad MF, Bennett PH. Diabetic kidney disease in Pima Indians. Diabetes Care 1993;16:335–41.
47. Bojestig M, Arnqvist HJ, Hermansson G, Karlberg BE, Ludvigsson J. Declining incidence of nephropathy in insulin-dependent diabetes mellitus. N Engl J Med 1994;330: 15–28.
48. Rossing P, Rossing K, Jacobsen P, Parving HH. Unchanged incidence of diabetic nephropathy in IDDM patients. Diabetes 1995;44:739–43.
49. Klein R. Vision disorders in diabetes. In: Diabetes in America. Bethesda: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 1995. p. 293.
50. Skyler JS. Microvascular complications: retinopathy and nephropathy. Endocrinol Metab Clin North Am 2001;30:833–56.
51. Prevent Blindness America. R&B legend Gladys Knight sings praises of early detection and management of diabetes. Schaumberg (IL): Prevent Blindness America; 2003.
52. Adamu GB, Geoffrey CO, Bala GS, Ibrahim SA, Sani SH, Tambaya MA. Relationship between random blood sugar and body mass index in an African population. Int J Diabetes & Metabolism 2006; 14: 144-5.
53. Jhanghorbani M, Hedley AJ, Jones RB, Gilmour WH. Is the association between glucose level and "all causes" and cardiovascular mortality risk dependent on body mass index? Med. J.Islamic Republic Iran 1992; 6:205-12.
54. Diaz VA, Mainous AG, Baker R, Carnemolla M, Majeed A. How does ethnicity affect the association between obesity and diabetes? Diabet Med. 2007; 24(11): 1199-204.
55. WHO Global InfoBase team. Surveillance of chronic diseases and risk factors: Country level data and comparable estimates. Geneva: World Health Organisation, 2005.
56. Pelletier DL, Rahn M. Trends in body mass index in developing countries. Food and Nutrition Bulletin 1998; 19(3): 223-39.
57. DeFronzo RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care 1991; 14(3):173-94.
58. Felber JP. From obesity to diabetes. Pathophysiological considerations. Int J Obes Relat Metab Disord 1992; 16(12):937-52.
59. Knowler WC, Pettitt DJ, Saad MF, Charles MA, Nelson RG, Howard BV, Bogardus C, Bennett PH. Obesity in the Pima Indians: its magnitude and relationship with diabetes. Am J Clin Nutr. 1991; 53(6 Suppl):1543S-51S.
60. Steppan CM, Bailey ST, Bhat S, Brown EJ, Banerjee RR, Wright CM, Patel HR, Ahima RS, Lazar MA. The hormone resistin links obesity to diabetes. Nature 2001; 409(6818):307-12.
61. Niswender KD, Magnuson MA. Obesity and the beta cell: lessons from leptin. J Clin Invest. 2007; 117(10): 2753–6.
62. Kadow aki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. J Clin Invest. 2006; 116(7): 1784–92.
63. Kahn SE, Hull RL, Utzschneider KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. Nature 2006; 444(7121):840-6.
64. Deurenberg P, Yap M. The assessment of obesity: methods for measuring body fat and global prevalence of obesity. Best Pract Res Clin Endocrinol Metab. 1999; 13(1): 1-11.
65. T.S. Sanal1, N. S. Nair2, P. Adhikari3. Factors associated with poor control of type 2 diabetes mellitus: A systematic review and Meta-analysis. Journal of Diabetology, October 2011; 3:1.
66. Jiang Y, Nie L, Jing C. Association of glycosylated hemoglobin A1c control with the complications in type 2 diabetic patiets. J South Med University 2008; 28: 2180-2182.
67. Michal S, Taylor TR, Sholmo V, Alexander L, Rina E, Asher E, et.al. Characteristics of diabetics with poorly glyceic control who achieve good control. JABFM 2008; 21: 490-496.
68. Panartotto D, Roberto A, Schumacher MV. Factors associated with glycemic control in type 2 diabetes. Rev Assoc Med Bras 2008; 54: 314-321.
69. Ahmed AT, Karter AJ, Warton EM, Doan JU, Weisner CM. The relationship between alcohol consumption and glycemic control among patients with diabetes: The Kaiser permanente Northern California Diabetes registry. J Gen Intern Med 2007;23: 275-282.