NAPATA COLLEGE

Faculty of medicine

Department of community

Batch (3)

Vascular complications of diabetic patients in Khartoum state in 2022.

This is submitted for the fulfillment of

The requirement of MBBS.

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الايـــــــــــــة

قال تعالى في محكم تنزيله

(...يَرْفَعِ اللَّهُ الَّذِينَ آَمَنُوا مِنْكُمْ وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ وَاللَّهُ بِمَا تَعْمَلُونَ خَبِيرٌ**)**

صدق الله العظيم

الآية 11 من سورة المجادلة

DEDICATION

To our mothers.

To our fathers .

To our teachers

Acknowledgement

We are so grateful acknowledge our research supervisor, DR. Isam Jalal Elden E. for his assistance and great advices throughout the research course that greatly improved the manuscript and helped us to overcome the obstacles . And we are so grateful thinks to those who work at Omdurman Hospital department of statistics

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Abstract

Introduction:

The global incidence and prevalence of diabetes mellitus has increased significantly. Patients with diabetes mellitus are at heightened risk of both adverse microvascular and cardiovascular events. The medical management of patients with diabetes mellitus mandates comprehensive risk factor modification and antiplatelet therapy.

Methods

Retrospective study was conducted among diabetic patients who developed vascular complication in Omdurman Hospital one of the oldest hospital in Sudan affiliated to the Sudanese Ministry of Health. In Omdurman locality. From 8/3/2022 to 11/20/2022. A sample random sampling methods was used Check list of 162 female and 162 male participants.

Results:

Vascular complications can occurs in both types of diabetes, and increase incidence of these complications differs from age and gender.

Our research reveal that {52.8%} of type two diabetic complications occur in female, and {47.2%} occur in male.

And in type one {47.2} in female and {52.8} occur in males.

Conclusion:

In our research reveal that incidence of vascular complications is high type2 in females and the occurrence of these complications is related mostly with type one diabetes in male.

مستخلص

مقدمة:

لقد زاد معدل الإصابة بمرض السكري وانتشاره على مستوى العالم بشكل ملحوظ. المرضى المصابون بداء السكري معرضون بشكل متزايد لخطر الإصابة بأمراض الأوعية الدموية الدقيقة والقلب والأوعية الدموية. تتطلب الإدارة الطبية لمرضى السكري تعديل عامل الخطر الشامل والعلاج المضاد للصفيحات.

الطريقة:

أجريت دراسة أسترجع الماضي على مرضى السكري الذين أصيبوا بمضاعفات في الأوعية الدموية في قسم الإحصاء بمستشفى أم درمان في الفترة من 2022/3/8 إلى 2022/20/11. تم استخدام عينة من طرق أخذ العينات العشوائية لجمع 324 مشاركًا. تم جمع البيانات باستخدام ملفات المرضى.

النتائج:

يمكن أن تحدث مضاعفات الأوعية الدموية في كلا النوعين من مرض السكري ، وتختلف زيادة حدوث هذه المضاعفات باختلاف العمر والجنس. يكشف بحثنا أن 85 (52.8٪) من مضاعفات السكري من النوع الثاني تحدث عند الإناث ، و 76 [47.2٪} تحدث عند الذكور. وفي النوع الأول 77 (47.2} عند الإناث و 86 (52.8) عند الذكور.

الاستنتاج:

في بحثنا كشف أن حدوث مضاعفات الأوعية الدموية مرتفع من النوع 2 عند الإناث ويرتبط حدوث هذه المضاعفات في الغالب بمرض السكري من النوع الأول عند الذكور.

Chapter one

Introduction

Introduction

Diabetes mellitus is not merely a disorder of carbohydrate metabolism, but a cause of vascular disease affecting nearly all blood vessel types and sizes. Indeed, vascular complications are responsible for most of the morbidity, hospitalizations, and death that occur in patients with diabetes mellitus. (1)

Background:

More than 29 million Americans, or nearly 10% of the United States population, have diabetes mellitus (2) the prevalence of diabetes mellitus increased significantly from 1980 to 2012 and associated closely with an increase in the number of overweight and obese persons. (3,4) Of >660 000 patients in the National Health Interview Survey, the prevalence of diabetes mellitus in the United States increased from 3.5 per 100 persons in 1990 to 8.3 per 100 persons in 2012.4 Those who were Hispanic or with a high school education or less had a significantly greater rate of developing diabetes mellitus. Notably, over just the last 4 years of the survey, the incidence of new diabetes mellitus decreased from a peak of 8.8 per 1000 persons to 7.1 per 1000 persons, but the prevalence remained stable at 8.3 per 100 persons. Similarly, in the Framingham study, incidence of diabetes mellitus, although markedly elevated compared with observations from the 1970s, has recently stabilized, despite the increasing population weight burden (.3) More recent work has implicated novel genetic associations and suggest future translational research targets in the understanding of this disease. (5, 6).

The increasing prevalence of diabetes mellitus extends beyond the United States and is a global phenomenon. The Global Burden of Metabolic Risk Factors of Chronic Diseases Collaborating Group estimates that the prevalence of diabetes mellitus increased from 153 million in 1980 to 347 million in 2008.7 The highest prevalence of diabetes mellitus is in Oceania, North Africa, the Middle East, and the Caribbean, each with an age-standardized prevalence of diabetes mellitus of 21% to 25% in men and 21% to 32% in women .(7) Improvement of economic conditions, better living standards, and adoption of the adverse lifestyle habits of wealthier nations has levied a cost in terms of disease prevalence. For example, in China, the prevalence of diabetes mellitus increased from 2.3% in 1994 to 9.7%

in 2008. The geographic distribution of diabetes mellitus in China closely follows the per capita gross regional product, with higher gross regional products associated with a higher prevalence of diabetes mellitus .(8) In addition, the prevalence of diabetes mellitus is growing faster in urban compared with rural settings. This pattern has been noted in West African populations (9) and India (10) as well. Thus, as economic development continues, it is likely that the global diabetes mellitus pandemic will worsen.

Along with a greater prevalence of diabetes mellitus comes a heightened risk of vascular disease, which affects the microvasculature, arteries, and veins. This review will discuss the impact of diabetes mellitus on these circulatory components, making clear the importance of vascular disease in diabetes mellitus.

Microvascular Disease

There are 3 major manifestations of microvascular disease, retinopathy, nephropathy, and neuropathy that will be reviewed.

Retinopathy

Microvascular disease is strongly associated with hyperglycemia. Over the range of chronic hyperglycemia commonly seen in practice, there is an 11-fold increase in retinopathy compared with a 2-fold increase in coronary artery disease.(11) Despite the importance of hyperglycemia, some patients may develop early evidence of retinopathy as long as 7 years before the development of frank type 2 diabetes mellitus, indicating a contribution of insulin resistance. In addition to severity of hyperglycemia and duration of diabetes mellitus, other factors associated with retinopathy include hypertension, smoking, and dyslipidemia. These and other pathophysiologic mechanisms, including insulin resistance and inflammation, may contribute to the microvascular disease process. (12)

The earliest histopathologic sign of diabetes mellitus–related retinopathy is a loss of sericites. Peristyles wrap around the arteriolar and capillary endothelial cells and participate in maintenance of capillary tone, growth, and resistance to damage from oxidative stress .(13,14) The disease is then marked by basement membrane thickening, endothelial cell permeability, and the formation of micro aneurysms. (15) Broadly, there are 2 types of

retinopathy, non-proliferative (background) and proliferative. In non-proliferative retinopathy, patients may develop dot hemorrhages, which are small hemorrhages in the middle of the retina surrounded by hard lipid exudates. Retinal edema also may be seen. Proliferative retinopathy is the development of neovascularization on the retina, which can be complicated by vitreous hemorrhage. These latter changes, without treatment, can lead to vision impairment.

In an analysis of the National Health and Nutrition Survey, the prevalence of retinopathy in the diabetic population was 28.5%, and 4.4% of the total had threatened loss of vision. Male sex, higher glycosylated hemoglobin levels, longer duration of diabetes mellitus, higher blood pressure, and use of insulin all were associated with developing retinopathy (.16) In a pooled analysis of 35 studies of diabetic people, collected from 1980 to 2008 from around the world, the prevalence among those 20 to 79 years old was 35% for any retinopathy, 7% for proliferative retinopathy, and 10% for vision threatening retinopathy. (17) Patients of African or Caribbean descent have higher rates of retinopathy compared with Caucasians or south Asians. (18) The presence of microvascular disease is also a marker of diffuse vascular disease. Diabetic patients with retinopathy have a higher rate of atherosclerosis than diabetic patients without retinopathy. (19)

Diabetic retinopathy is a leading cause of blindness in the United States. It was responsible for ≈8% of cases of legal blindness and 12% of all new cases of blindness in the United States each year in the last decade of the twentieth century (.20) However, new treatments have improved outcomes with a significantly reduced rate of severe visual impairment. Despite the increase in diabetes mellitus over the last few decades and a commensurate increase in the number of patients with diabetic retinopathy to ≈4 to 5 million people in the United States, the number of patients with diabetes mellitus with visual impairment has decreased from 26% in 1997 to ≈19% in 2011 (21) whereas the overall rate of visual impairment in the civilian population has remained stable at 9.3%.

Systemic medical therapy has played an important role for microvascular disease and will be discussed later. There are 2 treatments specific to the eye, which have reduced the progression to blindness. Two clinical trials, the Early Treatment Diabetic Retinopathy Study and the Diabetic Retinopathy Study, established macular and pan-retinal

photocoagulation as primary therapy for these 2 ocular complications. (22,23) More recently, the use of injected vascular endothelial growth factor antagonists have been shown to improve outcomes in proliferative retinopathy and have come into use. (24–27) The timing, use, and role of this therapy in relation to photocoagulation is not established and will depend on the results of clinical studies.

Nephropathy

The pathophysiology of nephropathy in diabetes mellitus bears many similarities to retinopathy, including the development of basement membrane thickening and micron aneurysm formation. In addition, glomerular hyper filtration is associated with expansion of the extracellular matrix and the progression of tubular and glomerular sclerosis. These changes cause albuminuria. Nephropathy is defined as the loss of >500 mg/d of protein. It is preceded by microalbuminuria, defined as a loss of 30 to 299 mg/d. (28)

Diabetic nephropathy is found in as many as 7% of type 2 diabetic patients at the time of their diabetes mellitus diagnosis. It occurs in ≤12% patients with type 1 diabetes mellitus by 7 years, (29) and as many as 25% of patients with type 2 diabetes mellitus have evidence of nephropathy by 10 years after the diagnosis is made.30 the prevalence is significantly worse in Asia. In a study of 5549 patients with type 2 diabetes mellitus across 103 medical centers in 10 Asian nations or regions, 40% had microalbuminuria and 19% had microalbuminuria. (31) One contributor may be poor risk factor control because <12% met blood pressure goal levels, and the mean Hgb A1C was 7.8%. In 2011 in the United States, nearly 50 000 patients with diabetes mellitus began treatment for renal failure and >225 000 required either dialysis or a kidney transplant. (21).

Neuropathy

The development of diabetic neuropathy is associated with vascular and nonvascular abnormalities. In addition to basement membrane thickening and peristyle loss, there is evidence of decreased capillary blood flow to C fibers, resulting in attenuated perfusion of the nerves and attendant endoneurial hypoxia. The neuropathy is characterized by axonal thickening and eventual loss of neurons. (32) The clinical manifestation of diabetic neuropathy can vary widely, although there are 2 major types. The most common is a

chronic, symmetrical, length-dependent sensorimotor polyneuropathy, which is associated with severity and duration of hyperglycemia. (33, 34) The pathophysiology of this subtype is similar to the other microvascular manifestations of diabetes mellitus. (35) Less common are polyneuropathies that develop at more unpredictable times during the course of diabetes mellitus that may not be symmetrical. The polyneuropathies commonly present with pain or autonomic symptoms and the course may be fluctuating. (36).

Problem statement

Increase Number of mortality and complications of other disease for patient who have diabetes more than non-diabetes

Justification:

Diabetes cause a lot of complications especially vascular complication which is very serious to patients may threat life of those patients

Objectives

General objectives

Vascular complication of diabetic patient in Khartoum state in 2022

Specific objectives

1. To determine vascular complications of diabetes
2. To Compare vascular complications occur in both types of diabetes
3. To identify which types of diabetes cause more vascular complications

Chapter two

Literature review

Literature review

Adults with diabetes have an annual mortality of about 5.4 %(double the rate for non-diabetic adults), and their life expectancy is decreased on average by 5-10 years. Although the increased death rate is mainly due to cardiovascular disease, deaths from non-cardiovascular causes are also increased. A diagnosis of diabetes immediately increases the risk of developing various clinical complications that are largely irreversible and due to microvascular or macro vascular disease. Duration of diabetes is an important factor in the pathogenesis of complications, but other risk factors—for example, hypertension, cigarette smoking, and hypercholesterolemia—interact with diabetes to affect the clinical course of microangiopathy (37)

Vascular complications are pivotal to the devastating effects of diabetes mellitus, and occur as a result of hyperglycemia engendered link between the disease and oxidative stress. In the diabetics, vascular tissues are damaged due to hyperglycemia-mediated free radicals, particularly through oxidation of essential biomolecules such as DNA, proteins and lipids in these tissues. Depending on the type of blood vessels affected, there are different categories of diabetic complications. The two major ones are micro vascular and macro vascular complications. The latter indicates damage to large blood vessels, while the former describes damage to small blood vessels. This mini-review provides some important information on vascular complications in the diabetics.(38) Term Complications

The vascular complications of diabetes are classified as either microvascular (retinopathy, nephropathy, and neuropathy) or macro vascular, which includes coronary artery, peripheral, and cerebral vascular disease. The microvascular complications can develop within 5 years of the onset of T1D, but infrequently develop before the onset of puberty. Clinically significant macro vascular complications are virtually never seen until adulthood.

Intensive glycemic control decreases the risk of microvascular disease, retinopathy, nephropathy and neuropathy, and macro vascular disease.89 In addition to hyperglycemia, several other modifiable risk factors contribute to and influence the risk of vascular complications. Use of tobacco considerably increases the risk of onset and progression of nephropathy and macro vascular disease.90 Hypertension, likewise, is associated with

increased risk and rate of progression of retinopathy, nephropathy, and macro vascular disease. Dyslipidemia contributes to the risk of macro vascular disease, nephropathy, and retinopathy. A family history of hypertension or nephropathy increases the risk of nephropathy.

Development of diabetic complications is insidious, but can usually be detected years before the patient evidences symptoms or organ function is impaired. Systematic screening can detect abnormality at an early stage when intervention to arrest, reverse, or retard the disease process will have the greatest impact. Diabetic retinopathy is rare before the onset of puberty or in patients who have had T1D for less than 5 years. Therefore, annual dilated retinal examinations should begin 3 to 5 years after diagnosis after the child is ≥10 years old.38 Temporary rapid progression of retinopathy may occur when metabolic control drastically improves; in these circumstances retinal examinations should be performed more frequently.

Renal disease is first detected by persistent albuminuria. After 5 years of diabetes and age ≥10 years, an annual screening measurement of urine albumin and creatinine concentrations should be performed to detect microalbuminuria. Several methods can be used to screen for microalbuminuria. The most convenient and, therefore, preferred method is to measure the albumin-to-creatinine ratio in a random spot urine specimen. First-void collections on arising in the morning avoid the confounding effect of increased albumin excretion induced by upright posture. Timed collections, either 24-hours or timed overnight, are more accurate but less convenient than spot samples. Albumin excretion is transiently elevated by hyperglycemia, exercise, and febrile illness. Because of marked day-to-day variability in albumin excretion, microalbuminuria should be confirmed in at least two of three collections over a 3- to 6-month period to establish the diagnosis of diabetic nephropathy before instituting treatment.91 In contrast to the recommendations for T1D in children, monitoring lipids, urinary albumin excretion, and screening eye examinations should begin at diagnosis in T2D.15

Although sensitive cardiovascular testing may detect subtle autonomic abnormalities in some adolescents with diabetes, they tend to be transient and are of unknown clinical importance. Neurologic and circulatory complications of diabetes are seldom clinically significant in the pediatric and adolescent population

Diabetic Retinopathy

Definition

Retinal vascular complication of diabetes mellitus; classified into non proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR).

Epidemiology

Leading cause of blindness in US population aged 20–64 years old.

Insulin-Dependent Diabetes (Type I)

Juvenile onset, usually occurs before 30 years of age; most patients are free of retinopathy during first 5 years after diagnosis; 95% of patients with insulin-dependent diabetes mellitus (IDDM) get DR after 15 years; 72% will develop PDR and 42% will develop clinically significant macular edema (CSME); severity worsens with increasing duration of diabetes mellitus.

Non-Insulin-Dependent Diabetes (Type II)

Adult onset, usually diagnosed after 30 years of age; more common form (90%) with optimal control without insulin; DR commonly exists at the time of diagnosis (60%) in non-insulin-dependent diabetes mellitus (NIDDM) with 3% having PDR or CSME at diagnosis of diabetes; 30% will have retinopathy in 5 years and 80% in 15 years. Risk of DR increases with hypertension, chronic hyperglycemia, renal disease, hyperlipidemia, and pregnancy.

Symptoms

Asymptomatic, may have decreased or fluctuating vision. Advanced retinopathy can lead to complete blindness.

Signs

Non proliferative Diabetic Retinopathy

Grading of NPDR and risk of progression to PDR depend on the amount and location of hard and soft exudates, intra retinal hemorrhages, micro aneurysms (MA), venous beading and loops, and intra retinal microvascular abnormalities (IRMA). Cotton-wool spots, dot and blot hemorrhages, posterior sub capsular cataracts, and induced myopia/hyperopia (from lens swelling due to high blood sugar) are common; may have macular edema, which can be clinically significant (CSME); usually bilateral

Chapter three

Methodology

Methodology:

Check list study was conducted among diabetic patients who development vascular complication in Omdurman Teaching Hospital is one of governmental hospital located in Omdurman near west shohader market,is east housh Khalifa ,south Omdurman children hospital and north busta market. from 8/3/2022 to 11/20/2022. A sample random sampling methods was used Data was collected using retrospectives checklist.

Study design

We are used retrospective study

Study Area:

Data was collected at Omdurman Hospital one of the oldest hospital in Sudan affiliated to the Sudanese Ministry of Health. In Omdurman locality.

Study Population

The study was done to these diseases both type one and type two, who developed vascular complications.

3.3.1 Inclusion Criteria

324 Diabetes patients (type one and two). Who developed vascular complications

Exclusion:

Was exclude not Sudanese people and Diabetic patients not have complication

3.4 Sample Size:

The sample size was calculated by the following formula at the confidence level of 95% and degree of precision 0.05

$$n = \frac{\mathrm{N}}{1 + Ne^2}$$

Description:

n = required sample size

e = margin of error at 5% (standard value of 0.05)

N=total population

3.5 Data Collection:

Data collected using check list

3.6 study duration:

This study was conducted between 8/3/2022 to 11/20/2022

3.7 Statistical Analysis:

Analysis was calculated by using SPSS

Ethical approval was obtained from ethical committee of NAPATA COLLAGE.

Chapter four

Statistical analysis:

Statistical analysis:

Data was coded and entered using SPSS program version 20. A suitable statistical test was used. Quantitative data was expressed as the mean-standard deviation (SD). Chi-squire Test was used to determine differences in complications and qualitative variables, Person's correlation test compare between 2 groups as regards quantitative variables. A value of $P<0.05$ was considered statistically significant.

RESULTS

Significant differences between the 2 groups to word high the mean of DM in type two with (2.0 ± 2.05) with (0.3) difference , this mean difference impose the increase in DM all over in type two than the type one group.

Although there is a significant differences to word age onset al over the two groups that enhance deal of DM to word type one group with the age.

Baseline Characteristics

A total of 324 patients completed the protocol (163 patients of diabetes (type one) and 161 of non-diabetes (type two).

The baseline characteristics of the study population are shown in (Table 1, 4). Significant differences were observed between the 2 groups with respect to demographic and clinical data, including age and gender (table 1).

Table 1: Comparison between type two group and type one group regarding age and gender

		Type two	Type one	Test value	P-value	Sig.
		No. = 161	**No. = 163**			
Age	Mean SD	55.76 ± 13.77	51.72 ± 12.11	2.801*	0.005	HS
	Range	19 – 87	22 – 80			
Sex	Female	85 (52.8%)	77 (47.2%)	73.604*	0.000	HS
	Male	76 (47.2%)	86 (52.8%)			

Table 2: Comparison between type two group and type one group regarding basic clinical data and medication

		Type two	Type one	Test value	P-value	Sig.
		No. = 161	**No. = 163**			
Age at onset	Mean± SD	26.01 ± 13.40	5.36 ± 3.20	19.126*	0.000	HS
	Range	11 – 56	0 – 10			
DM	Mean± SD	2.14 ± 0.53	1.18 ± 0.44	17.225*	0.000	HS
	Range	2 – 3	1 – 3			

Complications:

Participants determine alto of complications included in table(3), (Retinopathy, Nephropathy, Hypertension, Arrhythmia, Ischemia, DKA, MI, IHD, Renal failure, Erectile dysfunction, lower limb amputation, CHF, Numberless, Exophthalmos, Interdigital infection).

Table 3: Descriptive statistics of Complications

No		Frequency	Percent	Valid Percent	Cumulative Percent
1	Retinopathy	118	24.4	24.4	24.4
2	Nephropathy	108	22.3	22.3	45.0
3	Hypertension	146	30.2	30.2	75.2
4	Arrhythmia	4	.8	.8	76.0
5	Ischemia	3	.6	.6	76.7
6	DKA	40	8.3	8.3	84.9
7	MI	24	5.0	5.0	89.9
8	IHD	27	5.6	5.6	95.5
9	Erectile disjunction	2	.4	.4	97.5
10	lower limp amputation	2	.4	.4	97.9
11	CHF	3	.6	.6	98.6
12	Numbness	2	.4	.4	99.0
13	Exophthalmos	3	.6	.6	99.6
14	Interdigital infection	2	.4	.4	100.0
Total	484	100.0	100.0		

The most three common complication according to patient's answers were included in (Hypertension) with (30.2%), and (24.4%) for (Retinopathy), followed by (Nephropathy) (22.3%

Table 4: Complications differences

		Descriptive statistics	**Test value•**	**P-value**	**Sig.**
Complications	Mean SD	4.05 ± 3.23	1006.9	0.000	HS
	Range	1 – 15			

Significant differences observed when chi-squire test used to determine Complications.

DM:

Patients" DM duration distributed on three level (less than 10 concluded 46.3%, 10-25 with 41% and more than 25 12.75%, frequency in table (5) with 1.6 of mean with 0.67 Sd.

Table 5: Descriptive statistics of DM

Duration of DM							
		Frequency	**Percent**	**Mean**	**Mode**	**Std. D**	**Range**
Valid	Less than 10	150	46.3	1.66	2	0.69	2
	10-25	133	41.0				
	More than 25	41	12.7				
	Total	324	100.0				

Correlation of DM with other variables:

As it's shown in (tables 3, 4), DM at baseline of study in both groups significantly affected by age nor sex and age onset.

Which means DM symptoms were more in both group (fig 5, table 6). Positive correlation when there is increase in age on set there is an increase in DM and vice versa.

Table 6: Correlation of DM with age, in type one group

	DM	
	R	**P-value**
Age	0.150	0.056
Sex	0.060	0.448
Age onset	**0.362***	**0.000**

Table 7: Correlation of DM with age, Sex and Age onset in type two group

	DM	
	R	**P-value**
Age	0.151	0.056
Sex	0.016	0.845
Age onset	**0.187***	**0.017**

Correlation of DM among the sample

Used Regression test to determine correlation of DM (with age onset. as the result on table below positive (R) correlation gets (0.6) ranged in the medial level of correlation (3-7), when the impact of onset is (36%) on DM as it in (R^2)

ANOVA[a]								
Model		Sum of Squares	Df	Mean Square	F	Sig.	R	R^2
	Regression	2376 9.46	1	2376 9.4	185.700	.000	0.605	0.366
	Residual	4121 4.34	322	127.995				
	Total	6498 3.81	323					
a. Dependent Variable: age at onset								
b. Predictors: (Constant), Duration of DM								

Chapter five

Discussion

Discussion

A total of 324 patients completed the protocol (163 patients of diabetes (type one) and 161 of diabetes (type two).

Complications are reveals as following:

Hypertension reveal 146(30.2%) from all vascular complications we maintained in our research which is the most common cause vascular complication.

Retinopathy 188 (24.4%) from all vascular complications we maintained in our research which is second most complication.

Nephropathy 108(22, 3%) from all vascular complications we maintained in our research which is third most complication.

DKA, IHD, MI which cause (8.3%, 5.6%, 5%) respectively.

Erectile dysfunction, lower limp amputation, numbness, interdigital infection each cases (4.0%).

CHF, exophthalmos each causes (3.0%).

More than 29 million Americans, or nearly 10% of the United States population, have diabetes mellitus (2) the prevalence of diabetes mellitus increased significantly from 1980 to 2012 and associated closely with an increase in the number of overweight and obese persons. (3,4) Of >660 000 patients in the National Health Interview Survey, the prevalence of diabetes mellitus in the United States increased from 3.5 per 100 persons in 1990 to 8.3 per 100 persons in 2012.4 Those who were Hispanic or with a high school education or less had a significantly greater rate of developing diabetes mellitus. Notably, over just the last 4 years of the survey, the incidence of new diabetes mellitus decreased from a peak of 8.8 per 1000 persons to 7.1 per 1000 persons, but the prevalence remained stable at 8.3 per 100 persons. Similarly, in the Framingham study, incidence of diabetes mellitus, although markedly elevated compared with observations from the 1970s, has recently stabilized, despite the increasing population weight burden (.3) More recent work has implicated novel genetic associations and suggest future translational research targets in the understanding of this disease. (5, 6).

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million in 1980 to 347 million in 2008.7 The highest prevalence of diabetes mellitus is in Oceania, North Africa, the Middle East, and the Caribbean, each with an age-standardized prevalence of diabetes mellitus of 21% to 25% in men and 21% to 32% in women .(7) Improvement of economic conditions, better living standards, and adoption of the adverse lifestyle habits of wealthier nations has levied a cost in terms of disease prevalence. For example, in China, the prevalence of diabetes mellitus increased from 2.3% in 1994 to 9.7% in 2008. The geographic distribution of diabetes mellitus in China closely follows the per capita gross regional product, with higher gross regional products associated with a higher prevalence of diabetes mellitus .(8) In addition, the prevalence of diabetes mellitus is growing faster in urban compared with rural settings. This pattern has been noted in West African populations (9) and India (10) as well. Thus, as economic development continues, it is likely that the global diabetes mellitus pandemic will worsen.

Along with a greater prevalence of diabetes mellitus comes a heightened risk of vascular disease, which affects the microvasculature, arteries, and veins. This review will discuss the impact of diabetes mellitus on these circulatory components, making clear the importance of vascular disease in diabetes mellitus.

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The earliest histopathologic sign of diabetes mellitus–related retinopathy is a loss of sericites. Peristyles wrap around the arteriolar and capillary endothelial cells and participate in maintenance of capillary tone, growth, and resistance to damage from oxidative stress .(13,14) The disease is then marked by basement membrane thickening, endothelial cell permeability, and the formation of micro aneurysms. (15) Broadly, there are 2 types of retinopathy, non-proliferative (background) and proliferative. In non-proliferative retinopathy, patients may develop dot hemorrhages, which are small hemorrhages in the middle of the retina surrounded by hard lipid exudates. Retinal edema also may be seen. Proliferative retinopathy is the development of neovascularization on the retina, which can be complicated by vitreous hemorrhage. These latter changes, without treatment, can lead to vision impairment.

In an analysis of the National Health and Nutrition Survey, the prevalence of retinopathy in the diabetic population was 28.5%, and 4.4% of the total had threatened loss of vision. Male sex, higher glycosylated hemoglobin levels, longer duration of diabetes mellitus, higher blood pressure, and use of insulin all were associated with developing retinopathy (.16) In a pooled analysis of 35 studies of diabetic people, collected from 1980 to 2008 from around the world, the prevalence among those 20 to 79 years old was 35% for any retinopathy, 7% for proliferative retinopathy, and 10% for vision threatening retinopathy. (17) Patients of African or Caribbean descent have higher rates of retinopathy compared with Caucasians or south Asians. (18) The presence of microvascular disease is also a marker of diffuse

vascular disease. Diabetic patients with retinopathy have a higher rate of atherosclerosis than diabetic patients without retinopathy. (19)

Diabetic retinopathy is a leading cause of blindness in the United States. It was responsible for ≈8% of cases of legal blindness and 12% of all new cases of blindness in the United States each year in the last decade of the twentieth century (.20) However, new treatments have improved outcomes with a significantly reduced rate of severe visual impairment. Despite the increase in diabetes mellitus over the last few decades and a commensurate increase in the number of patients with diabetic retinopathy to ≈4 to 5 million people in the United States, the number of patients with diabetes mellitus with visual impairment has decreased from 26% in 1997 to ≈19% in 2011 (21) whereas the overall rate of visual impairment in the civilian population has remained stable at 9.3%.

Systemic medical therapy has played an important role for microvascular disease and will be discussed later. There are 2 treatments specific to the eye, which have reduced the progression to blindness. Two clinical trials, the Early Treatment Diabetic Retinopathy Study and the Diabetic Retinopathy Study, established macular and pan-retinal photocoagulation as primary therapy for these 2 ocular complications. (22,23) More recently, the use of injected vascular endothelial growth factor antagonists have been shown to improve outcomes in proliferative retinopathy and have come into use. (24–27) The timing, use, and role of this therapy in relation to photocoagulation is not established and will depend on the results of clinical studies.

Nephropathy

The pathophysiology of nephropathy in diabetes mellitus bears many similarities to retinopathy, including the development of basement membrane thickening and micron aneurysm formation. In addition, glomerular hyper filtration is associated with expansion of the extracellular matrix and the progression of tubular and glomerular sclerosis. These changes cause albuminuria. Nephropathy is defined as the loss of >500 mg/d of protein. It is preceded by microalbuminuria, defined as a loss of 30 to 299 mg/d. (28)

Diabetic nephropathy is found in as many as 7% of type 2 diabetic patients at the time of their diabetes mellitus diagnosis. It occurs in ≤12% patients with type 1 diabetes mellitus by 7 years, (29) and as many as 25% of patients with type 2 diabetes mellitus have evidence of nephropathy by 10 years after the diagnosis is made.30 the prevalence is significantly worse in Asia. In a study of 5549 patients with type 2 diabetes mellitus across 103 medical centers in 10 Asian nations or regions, 40% had microalbuminuria and 19% had microalbuminuria. (31) One contributor may be poor risk factor control because <12% met blood pressure goal levels, and the mean Hgb A1C was 7.8%. In 2011 in the United States, nearly 50 000 patients with diabetes mellitus began treatment for renal failure and >225 000 required either dialysis or a kidney transplant. (21).

Neuropathy

The development of diabetic neuropathy is associated with vascular and nonvascular abnormalities. In addition to basement membrane thickening and peristyle loss, there is evidence of decreased capillary blood flow to C fibers, resulting in attenuated perfusion of the nerves and attendant endoneurial hypoxia. The neuropathy is characterized by axonal thickening and eventual loss of neurons. (32) The clinical manifestation of diabetic neuropathy can vary widely, although there are 2 major types. The most common is a chronic, symmetrical, length-dependent sensorimotor polyneuropathy, which is associated with severity and duration of hyperglycemia. (33, 34) The pathophysiology of this subtype is similar to the other microvascular manifestations of diabetes mellitus. (35) Less common are polyneuropathies that develop at more unpredictable times during the course of diabetes mellitus that may not be symmetrical. The polyneuropathies commonly present with pain or autonomic symptoms and the course may be fluctuating. (36).

Adults with diabetes have an annual mortality of about 5.4 %(double the rate for non-diabetic adults), and their life expectancy is decreased on average by 5-10 years. Although the increased death rate is mainly due to cardiovascular disease, deaths from non-cardiovascular causes are also increased. A diagnosis of diabetes immediately increases the risk of developing various clinical complications that are largely irreversible and due to microvascular or macro vascular disease. Duration of diabetes is an important factor in the pathogenesis of complications, but other risk factors—for example, hypertension, cigarette

smoking, and hypercholesterolemia—interact with diabetes to affect the clinical course of microangiopathy (37)

Vascular complications are pivotal to the devastating effects of diabetes mellitus, and occur as a result of hyperglycemia engendered link between the disease and oxidative stress. In the diabetics, vascular tissues are damaged due to hyperglycemia-mediated free radicals, particularly through oxidation of essential biomolecules such as DNA, proteins and lipids in these tissues. Depending on the type of blood vessels affected, there are different categories of diabetic complications. The two major ones are micro vascular and macro vascular complications. The latter indicates damage to large blood vessels, while the former describes damage to small blood vessels. This mini-review provides some important information on vascular complications in the diabetics.(38)

Chapter six

Conclusion and recommendation

Conclusion and recommendations

Conclusion

Over all the occurrence of vascular complications among diabetes is very high in both types and its occurrence is strong related to gender and age.

High prevalence of vascular complications was observed in the Omdurman hospital. Simple, effective and easily available tools may suffice to screen for the complication facilitating early diagnosis and referral.

Recommendation

According to our rearch result We recommend the healthcare minister of sudan to macke mor center of DM in every district in order to all diabetic patients to monitor their illness at least monthly and follow all WHO recommends by make a commitment to managing diabetes, don't smoke keep your pressure and cholesterol under control ,take care of your teeth and consider daily aspirin and weight reduction, routine investigations and seeking for treatment to prevent complications. Health mesentery affords diabetes center and educate patients have diabetes how care their self .

Limitation of the study

Our study has the following limitations; first, the study was retrospective using check list and couldn't identify causality. Second, the study was conducted in only one hospital. Third, an obvious limitation of the review was the retrospective nature of the included studies, and with the majority being reported online (possible sampling bias). Again in the review, we have not used other databases such as CINAHL, EMBASE, PsycInfo, and COCHRANE.

Chapter seven

References

References

1. Tseng CH. Mortality and causes of death in a national sample of diabetic patients in Taiwan. Diabetes Care. 2004; 27:1605–1609.CrossrefMedlineGoogle Scholar

2. Centers for Disease Control. National Diabetes Statistics Report, 2014. Centers for Disease Control; 2015. Accessed October 5, 2015.Google Scholar

3. Abraham TM, Pencina KM, Pencina MJ, Fox CS. Trends in diabetes incidence: the Framingham Heart Study. Diabetes Care. 2015; 38:482–487. doi: 10.2337/dc14-1432.CrossrefMedlineGoogle Scholar

4.Geiss LS, Wang J, Cheng YJ, Thompson TJ, Barker L, Li Y, Albright AL, Gregg EW. Prevalence and incidence trends for diagnosed diabetes among adults aged 20 to 79 years, United States, 1980-2012.JAMA. 2014; 312:1218–1226. doi: 10.1001/jama.2014.11494.CrossrefMedlineGoogle Scholar

5.Knowles JW, Xie W, Zhang Z, et al; RISC (Relationship between Insulin Sensitivity and Cardiovascular Disease) Consortium; EUGENE2 (European Network on Functional Genomics of Type 2 Diabetes) Study; GUARDIAN (Genetics UndeRlying DIAbetes in HispaNics) Consortium; SAPPHIRe (Stanford Asian and Pacific Program for Hypertension and Insulin Resistance) Study. Identification and validation of N-acetyltransferase 2 as an insulin sensitivity gene.J Clin Invest. 2015; 125:1739–1751. doi: 10.1172/JCI74692.CrossrefMedlineGoogle Scholar

6. Hivert MF, Vassy JL, Meigs JB. Susceptibility to type 2 diabetes mellitus–from genes to prevention. Nat Rev Endocrinal. 2014; 10:198–205. doi: 10.1038/nrendo.2014.11.CrossrefMedlineGoogle Scholar

7.Danaei G, Fin cane MM, Lu Y, Singh GM, Cowan MJ, Paciorek CJ, Lin JK, Farzadfar F, Khang YH, Stevens GA, Rao M, Ali MK, Riley LM, Robinson CA, Ezzati M; Global Burden of Metabolic Risk Factors of Chronic Diseases Collaborating Group (Blood Glucose). National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and

816 epidemiological studies with 370 country-years and 2·7 million participants. Lancet. 2011;
817 378:31–40. doi: 10.1016/S0140-6736(11)60679-X.CrossrefMedlineGoogle Scholar

818 8.Zuo H, Shi Z, Hussain A. Prevalence, trends and risk factors for the diabetes epidemic in
819 China: a systematic review and meta-analysis. Diabetes Res Clin Pract. 2014; 104:63–72.
820 doi: 10.1016/j.diabres.2014.01.002.CrossrefMedlineGoogle Scholar

821 9.Abubakari AR, Lauder W, Jones MC, Kirk A, Agyemang C, Bhopal RS. Prevalence and
822 time trends in diabetes and physical inactivity among adult West African populations: the
823 epidemic has arrived. Public Health. 2009; 123:602–614. doi:
824 10.1016/j.puhe.2009.07.009.CrossrefMedlineGoogle Scholar

825 10.Jayawardena R, Ranasinghe P, Byrne NM, Soares MJ, Katulanda P, Hills AP.
826 Prevalence and trends of the diabetes epidemic in South Asia: a systematic review and
827 meta-analysis.BMC Public Health. 2012; 12:380. doi: 10.1186/1471-2458-12-
828 380.CrossrefMedlineGoogle Scholar

829 11.Stratton IM, Adler AI, Neil HA, Matthews DR, Manley SE, Cull CA, Hadden D, Turner
830 RC, Holman RR. Association of glycaemia with macrovascular and microvascular
831 complications of type 2 diabetes (UKPDS 35): prospective observational study.BMJ. 2000;
832 321:405–412.CrossrefMedlineGoogle Scholar

833 12.Nguyen TT, Wang JJ, Wong TY. Retinal vascular changes in pre-diabetes and
834 prehypertension: new findings and their research and clinical implications.Diabetes Care.
835 2007; 30:2708–2715. doi: 10.2337/dc07-0732.CrossrefMedlineGoogle Scholar

836 13.Arboleda-Velasquez JF, Valdez CN, Marko CK, D'Amore PA. From pathobiology to
837 the targeting of pericytes for the treatment of diabetic retinopathy.Curr Diab Rep. 2015;
838 15:573. doi: 10.1007/s11892-014-0573-2.CrossrefMedlineGoogle Scholar

839 14.Hammes HP, Lin J, Renner O, Shani M, Lundqvist A, Betsholtz C, Brownlee M,
840 Deutsch U. Pericytes and the pathogenesis of diabetic retinopathy.Diabetes. 2002; 51:3107–
841 3112.CrossrefMedlineGoogle Scholar

15.Tarr JM, Kaul K, Chopra M, Kohner EM, Chibber R. Pathophysiology of diabetic retinopathy.ISRN Ophthalmol. 2013; 2013:343560. doi: 10.1155/2013/343560.CrossrefMedlineGoogle Scholar

16.Zhang X, Saaddine JB, Chou CF, Cotch MF, Cheng YJ, Geiss LS, Gregg EW, Albright AL, Klein BE, Klein R. Prevalence of diabetic retinopathy in the United States, 2005-2008.JAMA. 2010; 304:649–656. doi: 10.1001/jama.2010.1111.CrossrefMedlineGoogle Scholar

17.Yau JW, Rogers SL, Kawasaki R, et al; Meta-Analysis for Eye Disease (META-EYE) Study Group. Global prevalence and major risk factors of diabetic retinopathy.Diabetes Care. 2012; 35:556–564. doi: 10.2337/dc11-1909.CrossrefMedlineGoogle Scholar

18.Sivaprasad S, Gupta B, Gulliford MC, Dodhia H, Mohamed M, Nagi D, Evans JR. Ethnic variations in the prevalence of diabetic retinopathy in people with diabetes attending screening in the United Kingdom (DRIVE UK).PLoS One. 2012; 7:e32182. doi: 10.1371/journal.pone.0032182.CrossrefMedlineGoogle Scholar

19.Chen YW, Wang YY, Zhao D, Yu CG, Xin Z, Cao X, Shi J, Yang GR, Yuan MX, Yang JK. High prevalence of lower extremity peripheral artery disease in type 2 diabetes patients with proliferative diabetic retinopathy.PLoS One. 2015; 10:e0122022. doi: 10.1371/journal.pone.0122022.MedlineGoogle Scholar

20.Centers for Disease Control and Prevention. Blindness caused by diabetes–Massachusetts, 1987–1994.MMWR Morb Mortal Wkly Rep. 1996; 45(43):937–941.Google Scholar

21.Centers for Disease ControlNational Diabetes Statistics Report, 2014. Centers for Disease Control and Prevention; 2015. Available from: http://www.cdc.gov/diabetes. Accessed October 7, 2015.Google Scholar

22.Photocoagulation treatment of proliferative diabetic retinopathy. Clinical application of Diabetic Retinopathy Study (DRS) findings, DRS Report Number 8. The Diabetic Retinopathy Study Research Group.Ophthalmology. 1981; 88(7):583–600.MedlineGoogle Scholar

870 23.Photocoagulation for diabetic macular edema. Early Treatment Diabetic Retinopathy
871 Study report number 1. Early Treatment Diabetic Retinopathy Study research group.Arch
872 Ophthalmol. 1985; 103(12):1796–806.CrossrefMedlineGoogle Scholar

873 24.Bressler SB, Qin H, Melia M, Bressler NM, Beck RW, Chan CK, Grover S, Miller DG;
874 Diabetic Retinopathy Clinical Research Network. Exploratory analysis of the effect of
875 intravitreal ranibizumab or triamcinolone on worsening of diabetic retinopathy in a
876 randomized clinical trial.JAMA Ophthalmol. 2013; 131:1033–1040. doi:
877 10.1001/jamaophthalmol.2013.4154.CrossrefMedlineGoogle Scholar

878 25.Diabetic Retinopathy Clinical Research Network. Randomized clinical trial evaluating
879 intravitreal ranibizumab or saline for vitreous hemorrhage from proliferative diabetic
880 retinopathy.JAMA Ophthalmol. 2013; 131:283–293. doi:
881 10.1001/jamaophthalmol.2013.2015.

882 26.Bhavsar AR, Torres K, Glassman AR, Jampol LM, Kinyoun JL; Diabetic Retinopathy
883 Clinical Research Network. Evaluation of results 1 year following short-term use of
884 ranibizumab for vitreous hemorrhage due to proliferative diabetic retinopathy.JAMA
885 Ophthalmol. 2014; 132:889–890. doi:
886 10.1001/jamaophthalmol.2014.287.CrossrefMedlineGoogle Scholar

887 27.Mirshahi A, Roohipoor R, Lashay A, Mohammadi SF, Abdoallahi A, Faghihi H.
888 Bevacizumab-augmented retinal laser photocoagulation in proliferative diabetic retinopathy:
889 a randomized double-masked clinical trial.Eur J Ophthalmol. 2008; 18:263–
890 269.CrossrefMedlineGoogle Scholar

891 28.Consensus development conference on the diagnosis and management of nephropathy in
892 patients with diabetes mellitus. American Diabetes Association and the National Kidney
893 Foundation.Diabetes Care. 1994; 17(11):1357–1361.CrossrefMedlineGoogle Scholar

894 29.Chaturvedi N, Bandinelli S, Mangili R, Penno G, Rottiers RE, Fuller JH.
895 Microalbuminuria in type 1 diabetes: rates, risk factors and glycemic threshold.Kidney Int.
896 2001; 60:219–227. doi: 10.1046/j.1523-1755.2001.00789.x.CrossrefMedlineGoogle
897 Scholar

30.Adler AI, Stevens RJ, Manley SE, Bilous RW, Cull CA, Holman RR; UKPDS GROUP. Development and progression of nephropathy in type 2 diabetes: the United Kingdom Prospective Diabetes Study (UKPDS 64).Kidney Int. 2003; 63:225–232. doi: 10.1046/j.1523-1755.2003.00712.x.CrossrefMedlineGoogle Scholar

31.Wu AY, Kong NC, de Leon FA, Pan CY, Tai TY, Yeung VT, Yoo SJ, Rouillon A, Weir MR. An alarmingly high prevalence of diabetic nephropathy in Asian type 2 diabetic patients: the MicroAlbuminuria Prevalence (MAP) Study.Diabetologia. 2005; 48:17–26. doi: 10.1007/s00125-004-1599-9.CrossrefMedlineGoogle Scholar

32.Tavakoli M, Mojaddidi M, Fadavi H, Malik RA. Pathophysiology and treatment of painful diabetic neuropathy.Curr Pain Headache Rep. 2008; 12:192–197.CrossrefMedlineGoogle Scholar

33.Dyck PJ, Davies JL, Clark VM, Litchy WJ, Dyck PJ, Klein CJ, Rizza RA, Pach JM, Klein R, Larson TS, Melton LJ, O'Brien PC. Modeling chronic glycemic exposure variables as correlates and predictors of microvascular complications of diabetes.Diabetes Care. 2006; 29:2282–2288. doi: 10.2337/dc06-0525.CrossrefMedlineGoogle Scholar

34.Dyck PJ, Davies JL, Wilson DM, Service FJ, Melton LJ, O'Brien PC. Risk factors for severity of diabetic polyneuropathy: intensive longitudinal assessment of the Rochester Diabetic Neuropathy Study cohort.Diabetes Care. 1999; 22:1479–1486.CrossrefMedlineGoogle Scholar

35.Giannini C, Dyck PJ. Ultrastructural morphometric abnormalities of sural nerve endoneurial microvessels in diabetes mellitus.Ann Neurol. 1994; 36:408–415. doi: 10.1002/ana.410360312.CrossrefMedlineGoogle Scholar

36.Tesfaye S, Boulton AJ, Dyck PJ, Freeman R, Horowitz M, Kempler P, Lauria G, Malik RA, Spallone V, Vinik A, Bernardi L, Valensi P; Toronto Diabetic Neuropathy Expert Group. Diabetic neuropathies: update on definitions, diagnostic criteria, estimation of severity, and treatments. Diabetes Care. 2010; 33:2285–2293. doi: 10.2337/dc10-1303.

37.Richard Donnelly, Alistair M Emslie-Smith, Iain D Gardner, Andrew D 38.MorriBmj 320 (7241), 1062-1066, 2000 pubmed

Annexes:

The city is located in heart of Sudan at confluence of the White Nile and Blue Nile where the two rivers unite to form the river Nile. The confluence of the two rivers creates a unique effect.as they join each river retains its own color. These colors are more visible in the flood season.

The state lies between longitudes 31.5 to 34E and latitudes 15 to 16 N . it is surrounded by river Nile state in the North –east , in the east and southeast by the estates of Kassala ,Qadarif and white Nile state and in the west by North Kurdufan.


RB-DESKKART
ÄGYPTEN
LIBYEN
TSCHAD
SÜDSUDAN
ERITREA
ÄTHIOPIEN
Rotes Meer
Nasser-Stausee
Wadi Halfa
Amara West
Halaib
Jebel Is 1851 m
Jebel Asoteriba 2216 m
Ras Abu Shagara
Jebel Erba 2213 m
Muhammad Qol
Jebel Oda 2259 m
Port Sudan
Suakin
1573 m
1757 m
Karora
Jebel Hamoyet 2780 m
Nubische Wüste
Abu Hamed
Libysche Wüste
Merga-Oase
Dongola
Heiliger Berg Jebel Barkal
Merowe
Old Dongola
Ed Debba
Napata-Region
Atbara
Pyramiden von Meroë
Ed Damer
Nil
Atbara
Khartoum-Nord
Omdurman
KHARTOUM
Kassala
Blauer Nil
Wad Medani
Gedaref
Darfur
Jebel Teljo 1954 m
Jebel Mun 1309 m
Jebel Gurgei 2397 m
Sodiri
Ed Dueim
Sennar
Rahad
Dinder
Gallabat
Marra-Plateau
El Geneina
El Fasher
Wad Banda
El-Obeid
Rabak
Singa
Jebel Marra 3088 m
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Kordofan
Nyala
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SUDAN

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