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DESCRIPTION OF DIABETES, RISK FACTORS, ADVANCEMENTS IN DIABETES TREATMENT, AND THE EFFECT OF DIET ON QUALITY OF LIFE

Dr. R Gautham Chakra*[1], Sk. Suhana[2], J. Naveen Kumar[2], V. Krishna Devi[2], G. Ulitappa[2], B. Hasini[2], K. Mahesh[2], A. Pradeep[2], S. Ashraf Ali[2]

[1]Department of Pharmacy Practice, Saastra College of Pharmaceutical Education & Research, Jwalamukhi temple, Varigonda, Totapalli Gudur mandal, near Varigonda, Nellore, Andhra Pradesh 524311 India.

[2]Saastra College of Pharmaceutical Education & Research, Jwalamukhi temple, Varigonda, Totapalli gudur Mandal, near Varigonda, Nellore, Andhra Pradesh 524311 India.

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Abstract

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia due to abnormalities in insulin production, activity, or both. This condition leads to long-term complications, including dysfunction and damage to the kidneys, heart, nerves, eyes, and blood vessels. Insulin resistance, particularly in skeletal muscles, adipose tissue, and the liver, plays a key role in the metabolic defects associated with diabetes. A prospective study was conducted at KIMS Hospital's General Medicine Department to assess the development of diabetic medications. Sixty patients were included, and the study summarized the development of insulin, oral drugs, and injectable non-insulin drugs for treating hypoglycemia. The pharmacological properties and side effects were briefly reviewed SGLT2 inhibitors, DPP-4 inhibitors, and GPR40 agonists are promising treatments. Future research should focus on understanding the mechanisms of diabetes, preventative measures, early diagnosis, and developing new drugs with fewer side effects.

∗Corresponding Author
Name: Dr. R Gautham Chakra
Phone: +91 7674016126
Email: gauthamrowdhra05@gmail.com

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INTRODUCTION

Hyperglycemia, a collection of metabolic conditions known as diabetes mellitus, is caused by insulin action, secretion, or both deficiencies. Over time, hyperglycemia can cause several organs, including the kidney, heart, nerves, eyes, and blood vessels, to fail, malfunction, or be destroyed [1]. Metabolic defects are caused by reduced insulin synthesis to achieve the proper response and insulin resistance in particular tissues, primarily the liver, adipose tissue, and skeletal muscles at the level of effector enzymes, signal transduction system, or Insulin receptors.

Genes are also responsible for these abnormalities. The assessment of hyperglycemia involves multiple morbidities [2]. These include malformations that lead to an inability to respond to Insulin and autoimmune destruction of the pancreatic beta cells, which results in insufficient Insulin. Insufficient insulin activity results from either inadequate insulin synthesis or a reduction in the tissue's response to Insulin, which causes abnormalities in the metabolism of proteins, fats, and carbs in hyperglycemia. The severity of the symptoms varies according to how long the hyperglycemia lasts [3]. Some hyperglycemia sufferers have fewer symptoms, particularly for those with type-2 hyperglycemia in the early stages of the illness. Polyurea, polyphagia, polydipsia, weight loss, and hazy vision are among the symptoms. If left unchecked, uncontrolled hyperglycemia can cause stupor, coma, and even death from ketoacidosis or, in rare cases, nonketotic hyperosmolar syndrome[4]. Retinopathy may result in blindness, neuropathy, which can harm the kidneys, and peripheral neuropathy, which causes foot ulcers and disseminates diabetic neuropathy. Autonomic neuropathy, which causes gastrointestinal, urogenital, cardiovascular, peripheral artery, and cerebrovascular diseases, is an example of the long-term consequences of hyperglycemia. Patients with hyperglycemia typically have high blood pressure and low lipoprotein levels [5].

MATERIALS AND METHODS:

Method and collection of data:

Site of Study:

The Study of **"Description of diabetes, risk factors, advancements in diabetes treatment, and the effect of diet on the quality of life."** This was carried out at Krishna Institute of Medical Sciences, Nellore. Under the guidance of Dr. R. Gautam Chakra, Assistant Professor, Saastra College of Pharmacy, Nellore.

Study Design:

This observational study is being carried out prospectively in the outpatient departments [6].

Study Period:

A six-month prospective observational study was conducted at the Krishna Institute of Medical Sciences in Nellore.

Study site:

The study will be conducted in the Kims Hospital, Nellore.

Study population:

Approximately 100 patients who were diagnosed were presently under treatment.

Study duration:

6 months (February 2024 to July 2024).

Source of data:

Patients' case sheets, blood samples, and interviews were also included.

Patient selection criteria:

The patients enrolled in the study were selected based on inclusion and exclusion criteria [7].

Inclusion criteria:

- Diabetic. Patients with hypertension and kidney comorbidities taking meglitinide are included.
- Patients who agree to participate in the study are included.
- Patients aged >35 to <90 years are included.

Exclusion criteria:

- Patients under 20 years old are excluded.
- Excludes pregnant and lactating mothers.
- Insulin-treated patients are excluded.
- Patients with COVID-19 are excluded [8].
- Patients who do not take meglitinide are excluded.
- Patients who are unwilling to engage in the trial will be excluded.
- Patients with CNS or hepatic impairment are excluded.
- Eligible patients will provide informed permission for the trial [9].

Study procedure:

1. The Institutional Ethics Committee of Saastra College of Pharmacy, Nellore, and the Department of General Medicine, KIMS Hospital, Nellore, approved the study, and a uniform data entry format was created for gathering patient details.
2. The study's eligible participants provided data, including demographics, a history of comorbidities between diabetes and

hypertension, the use of meglitinides, and social history in patients with diabetes.

3. The patient will have a three-month follow-up to determine the prognosis at the start of treatment.
4. Meglitinide's impact on patients with diabetic kidney damage will be tracked, and information will be gathered [10].
5. A questionnaire regarding social history, smoking, drinking, and other habits will be used to gather information.
6. Forms for patient counseling were provided.
7. Patients receive counseling regarding the use of meglitinide and lifestyle changes with their complaints at each follow-up visit.
8. Patients are monitored continuously, the effects of meglitinide are seen, and the patient's Quality of life is assessed.
9. Formatting the results, sending them in, and publishing the data in suitable journals with high impact and indexing [11].

Statistical analysis:

The measurement data were analyzed using the Statistical Package for the Social Sciences (SPSS) software, and the results are presented as the mean ± standard deviation. The Student's t-test was used to analyse group comparisons. The $\chi 2$ test was used to compare sample rates in measurement data, which are expressed as percentages. A difference that was deemed statistically significant was defined as $P<0.05$ [12].

RESULTS AND DISCUSSION

RESULTS:

1. BASED ON GENDER:

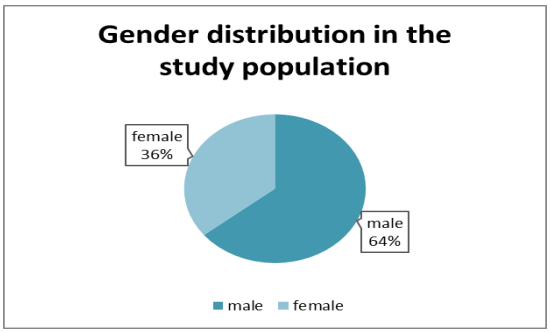

Figure 1 : Based on Gender

Table 1 Based on Gender

GENDER	NO OF PATIENTS	PERCENTAGE
Male	64	64.20%
Female	36	35.80%

2. AGE-BASED:

Table 2 Based on Age

Age	No. of Patients	Percentage
25-35	4	4.00%
35-45	12	12.00%
45-55	23	24.00%
55-65	34	35.00%
65-75	17	17.00%
75-85	8	8.00%

3. Based on Common Comorbidity:

Table 3 Based on Common Comorbidity

COMMON COMORBIDITY	NO OF PATIENTS
HYPOGLYCEMIA	2
UTI	11
RTI	2
CAD	10
CVA	3
HYPOTHYROIDISM	16
CKD	15
HTN	76

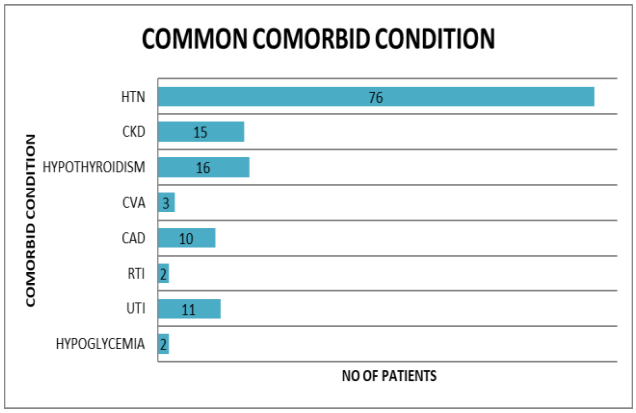

Figure 2 Based on Common Comorbidities

4. COMMON ORAL HYPOGLYCEMICS-BASED:

Table 4 Based on Common Oral Hypoglycemics

DRUGS	NO OF PATIENTS
Metformin	43
Glimepiride	9
Sitagliptin	2
Vildagliptin	1
Metformin +Glimepiride	4

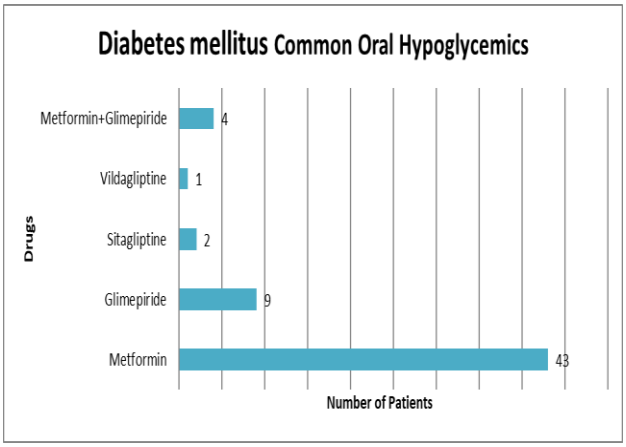

Figure 3 Common Oral Hypoglycemics Based

5. BASED ON THE TYPES OF INSULIN USED:

Table 5 : Based on types of Insulin used in Type 2 DM

INSULIN	NO OF PATIENTS
linctus	1
Human Mixtard insulin	25
Human Actrapid insulin	15
HMI+HAI	2

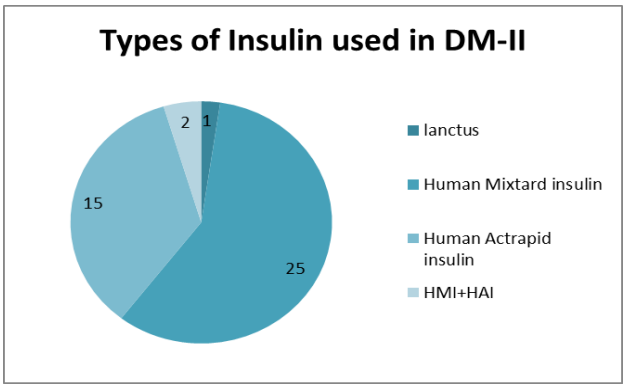

Figure 4 : Based on types of Insulin used in Type 2 DM

6. BASES ON SEVERITY OF DRUG INTERACTION:

Table 6 Based on Severity of Drug Interaction

SEVERITY	NO OF CASES
SIGNIFICANT	53
SERIOUS	10
NO	25
MINOR	10

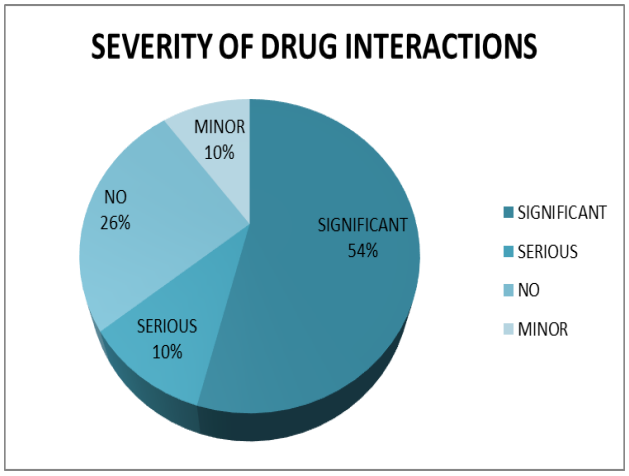

Figure 5 Based on Severity of Drug Interaction

DISCUSSION:

Males (64.20%) and females (34.80%) made up the gender distribution in the study. The age distribution was determined to be 25–35 years (4%), 35–45 years (12%), 45–55 years (54%), 55–65 years (35%), 65–75 years (17%), and 75–85 years (8%). Comorbid illnesses were hypertension (76), hypothyroidism (16), chronic renal disease (15), urinary tract infections (11), and coronary artery disease (10), out of the 98 cases that were recruited.

During the trial, 464 prescription drugs were written. Among the prescription drugs were 102 (22%) anti-diabetics, 72 (15.5%) antihypertensives, 59 (12.72%) multivitamins, 46 (9.9%) antiplatelets, 20 (4.31%) statins, and 165 (35.5%) other drugs.

The antidiabetics that were prescribed were metformin for 44 patients (43.14%), glimepiride for nine patients (8.83%), sitagliptin for two patients (1.96%), vildagliptin for one patient (0.98%), and Insulin for 43 patients (42.16%).

Table 7 Case Study-1 Hematology and Coagulation Test

ANALYTE	TEST	REFERENCE	UNITS
HEMATOLOGY & COAGULATION			
CBC			
RBC	4	M – 4.5-5.5 F – 3.8-4.8	Milli./Cu mm
Hb	10	M – 13-17 F - 12-15	g/dl
PCV	38	M – 41-49 F – 37-46	%
WBC		4000-11000	Cells/cu mm
DC N L E B M		$N_{40-70\%}$ $L_{20-40\%}$ $E^{1-6\%}$ $B_{<1\%}$ $M_{2-10\%}$	%
MCV		76-96	FI
MCH		27-32	Ng
MCHC		31-35	g/l
ESR		3-15	mm/Hr
PLATELET COUNT		1.5-4.0	Lakhs/cu mm
COAGULATION PROFILE			
BT		2-6	Mint.
CT		3-10	Mint.
PT		11-16	Sec.
PTT		30-40	Sec.
GLYCEMIC PROFILE			
RBS	90	80-140	Mg/dl
FBS	142	70-110	
PPBS	258	80-140	
PLASMA INSULIN (BIOASSAY)		11-240	µIU/ml
PROINSULIN		2-26	Pmol/lit
GLYCATED HEMOGLOBIN (HbA1C)	8.2	< 8%	%
RENAL FUNCTION TESTS			
UREA	2.8	2.4 – 4.8	mmol/L
UREA NITROGEN		3 - 9	
BUN (UREA)		7 - 18	mg/dl
CREATININE (C_T)	0.8	0.6 – 1.4	
ELECTROLYTES			
SODIUM (Na^+)	136	134-144	mmol/L
Potassium (K^+)	3.2	3.5-5.0	
CHLORIDE (CI)	98	98-106	
BICARBONATE ($HC0_3$)		21-28	
MINERALS			
Calcium (Ca^{2+})	9	8-6-103	mg/dl

Table 7 Case Study-1 Hematology and Coagulation Test (Continued)

Phosphorus		2.5-4.8 1.6-2.6 35-140	
Magnesium (Mg^{+})			
Iron			Mcg/dl
TIBC		245-400	
LIPID PROFILE			
Total Cholesterol	160	<200	Mg/dl
Triglycerides		<150	
HDL cholesterol	35	>40	
LDL cholesterol		<100	
LIVER FUNCTION TESTS			
Total Bilirubin		0.3-1.2	Mg/dl
Direct Bilirubin		0.1-0.3	
Indirect Bilirubin		0.1-0.5	
SGPT (ALT)		30-65	U/L
SGOT (AST)		15-37	
TOTAL PROTEIN		6.4-8.2	g/dl
ALBUMIN		3.5-5.2	
GLOBULIN		2-3.5	
A/G ratio		1.5-3:1	
Alkaline Phosphate		33-96	U/L
CARDIAC P PROFILE			
CPK – MB		2-24	U/L
LDH		207-414	
CPK		38-174	
THYROID PROFILE			
FT3		2.3-4.2	Pg/ml
FT4		0.8-1.8	Ng/dl
TSH		0.35-5.50	mIU / ml
FSH		9-15	

Metformin plus glimepride was the most often given FDC (4, 3.9%).

Additionally, 5.1% of patients received clopidogrel and aspirin, and 4.1% of IHD patients received aspirin. Statins were prescribed to every dyslipidemia patient.

Patients with uncontrolled diabetes were prescribed more clopidogrel, although aspirin was prescribed more in patients with managed diabetes.

Among the patients with managed diabetes, CCB prescriptions increased. Patients with uncontrolled diabetes used combination antihypertensive drugs more often than those with controlled diabetes. The only individuals who received AT1 receptor blockers were those with uncontrolled diabetes. The more significant number of uncontrolled diabetic patients may be a result of their lack of knowledge, poor adherence to treatment, and ignorance. As a result, additional medications or drug combinations can be required to treat their coexisting illnesses. The drug interactions that were discovered were categorized according to their severity. Of the patients in the study group, 25 had no interactions, 10 had severe interactions, 53 had significant interactions, and 10 had minor interactions.

CASE STUDY-1:

Patient Name: Mrs. Pooja

Age/Gender: SS/F

Department/Ward/Unit: General Medicine

Name of the Consultant Doctor/Physician: Dr. Naresh Kumar

Past Medical History: Nill

Past Medication History: Nill

Allergies (Drug/Food/Other): Dust allergy

Personal/Social history and habits:

Height/Weight: 156cm / 80kgs

Alcoholic/Smoker: No

Diet (Veg/non-veg): Mixed

Sleep: 6-8 hrs sleep

Bowel and Bladder habit: Regular

Exercise: No

Family history: No known cause of HTN and DM

General Examination: Patient is conscious and coherent

Physical Examination: Vital signs:

Temperature (^{0}F): 98

Pulse rate (/mint): 87/min

Respiratory rate (beats/mint): 19bpm

Blood Pressure (mm of Hg): 130/80

Systems Examination:

CVS: S1S2+

RS: BAE+

CNS: NFND

GI (ABDOMEN) and GU: BMI – 32.87

Dermatologist (Skin): Normal

Upper and Lower Limbs: Weakness in the right limb

LAB INVESTIGATION:

URINE ANALYSIS:

COLOR: Normal

SPECIFIC GRAVITY:

pH:

ALBUMIN: 2.2

SUGAR:

KETONE: 3.6

BLOOD:

PUS CELLS:

EPITHELIAL CELLS:

RBC:

Other investigations (X-ray, CT, US & any other): FBG, PPG, HbA1C

CONFORMATION DIAGNOSIS: Type- 2 DM with Obesity (BMI – 32.87)

TREATMENT CHART: T. Metformin 500mg - BID

CASE STUDY-2:

Patient Name: Mrs. P. Sumathi

Age/Gender: 48 years/Male

Admission (IP) No: IP18034

Department/Ward/Unit: Neurology

Name of the Consultant Doctor/Physician: Dr. Vinay

Provisional/Admitting diagnosis: Ischemic stroke, Right hemiparesis Hypertension, and Type II DM

Chief Complaints: Right upper and lower limb weakness, Mouth deviation, Headache, Giddiness, Slurred speech

Past Medical History: Type II DM

Past Medication History: Nill

Allergies (Drug/Food/Other): Nill

Personal /Social history and habits:

Height/Weight: 5.6/62kgs

Appetite: Normal

Education: Not specified

Alcoholic/smoker: Nill

Diet (veg/non-veg): Mixed

Sleep: Normal

Bowel & Bladder habit: Normal

Exercise:

Family History: Not Significant

GENERAL EXAMINATION:

Physical Examination: Vital signs

Table 8 Case Study-2 Hematology and Coagulation Test

ANALYTE	TEST	REFERENCE	UNITS
HEMATOLOGY & COAGULATION			
CBC			
RBC		M – 4.5-5.5 F – 3.8-4.8	Milli./Cu mm
Hb	12.5	M – 13-17 F - 12-15	g/dl
PCV		M – 41-49 F – 37-46	%
WBC	10,600	4000-11000	Cells/cu mm
DC N L E B M	25% 04% 00% 05%	$N_{40-70\%}$ $L_{20-40\%}$ $E^{1-6\%}$ $B_{<1\%}$ $M_{2-10\%}$	%
MCV		76-96	FI
MCH		27-32	Ng
MCHC		31-35	g/l
ESR		3-15	mm/Hr
PLATELET COUNT	2.65	1.5-4.0	Lakhs/cu mm
COAGULATION PROFILE			
BT		2-6	Mint.
CT		3-10	Mint.
PT		11-16	Sec.
PTT		30-40	Sec.
GLYCEMIC PROFILE			
RBS		80-140	Mg/dl
FBS		70-110	
PPBS		80-140	
PLASMA INSULIN (BIOASSAY)		11-240	µIU/ml
PROINSULIN		2-26	Pmol/lit
GLYCATED HEMOGLOBIN (HbA1C)		< 8%	%
RENAL FUNCTION TESTS			
UREA		2.4 – 4.8	mmol/L
UREA NITROGEN		3 - 9	
BUN (UREA)		7 - 18	mg/dl
CREATININE (C_T)	0.9	0.6 – 1.4	
ELECTROLYTES			
SODIUM (Na^+)	136	134-144	mmol/L
Potassium (K^+)	4.2	3.5-5.0	
CHLORIDE (CI)		98-106	
BICARBONATE ($HC0_3$)		21-28	
MINERALS			
Calcium (Ca^{2+})	9	8-6-103	mg/dl
Phosphorus		2.5-4.8 1.6-2.6 35-140	

Table 9 Case Study-2 Hematology and Coagulation Test (continued)

Magnesium (Mg^{+})			
Iron			Mcg/dl
TIBC		245-400	
LIVER FUNCTION TESTS			
Total Bilirubin		0.3-1.2	Mg/dl
Direct Bilirubin		0.1-0.3	
Indirect Bilirubin		0.1-0.5	
SGPT (ALT)		30-65	U/L
SGOT (AST)		15-37	
TOTAL PROTEIN		6.4-8.2	g/dl
ALBUMIN		3.5-5.2	
GLOBULIN		2-3.5	
A/G ratio		1.5-3:1	
Alkaline Phosphate		33-96	U/L
CARDIAC P PROFILE			
CPK – MB		2-24	U/L
LDH		207-414	
CPK		38-174	
THYROID PROFILE			
FT3		2.3-4.2	Pg/ml
FT4		0.8-1.8	Ng/dl
TSH		0.35-5.50	mIU / ml
FSH		9-15	

Temperature (^{0}F): 98.6 F

Pulse rate (/min.): 119 bpm

Respiratory rate (beats/min.): 21/min

Blood Pressure (mm of Hg): 160/90 mm of HG

SYSTEMS EXAMINATION:

CVS: S1S2+

RS: BLAE+

CNS: E4V5M6

GI (ABDOMEN) & GU: Normal

Upper & Lower Limbs

URINE ANALYSIS

COLOR: Normal

SPECIFIC GRAVITY:

pH:

ALBUMIN: 2.2

SUGAR:

KETONE: 3.6

BLOOD:

PUS CELLS:

EPITHELIAL CELLS:

RBC:

Other investigations (X-ray, CT, US & any other): MRI BRAIN: Acute infarct in left half of the pons and left half of the medulla persistent right fetal PCA with mild narrowing of Left MCA

2D ECHO: No RMWA, Good LV Function, Grade I DD. EF-63%

CONFORMATION DIAGNOSIS: Ischemic stroke, Right hemiparesis, and Acute infarcts in the left half of pons and left half of medulla and Type II DM

FOLLOW UP MEDICINE:

Tab. Cutin	80 mg	OD	0---0---1
Tab.Biohomin		OD	0---0---1
Tab. Preva-AS		OD	0---1---0
Tab.Megamol-P			SOS
Tab.Telzoe-AM	40mg	OD	½--0—0

Tab.Stemitil-MD BD 1---0---1

Tab.Arthopan

DIABETES MEDICATION:

Tab. Glimirep MV 2.2 BD 1---0---1

CONCLUSION:

Finding the best treatment for DM becomes a primary priority in the fight against the disease as its occurrence rises. Several treatment approaches have been established, including GPR40 agonists, DPP-4, and SGLT2 inhibitors. To treat diabetes mellitus (DM) and prolong life, more research should be done on the following topics: (1) the precise mechanism causing the disease and its associated complications; (2) successful intervention trials and preventative measures to prevent the occurrence of this disease; (3) early diagnosis for earlier treatment; and (4) novel drugs with more beneficial effects and less adverse effects.

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Conflict of Interest

The authors declare no conflict of interest, financial or otherwise.

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