ORIGINAL ARTICLE

INTERNATIONAL JOURNAL OF EXPERIMENTAL AND BIOMEDICAL RESEARCH

Published by Pharma Springs Publication Journal Home Page: https://pharmasprings.com/ijebr

A Tertiary Care Hospital's Prescription Pattern for Diabetes Mellitus

Gautham Chakra R*[1], Rajeswari G[2], Alekya B[2], Mahaboob Ali Sk[2], Noorul Kareem M, Prekshasri S[2]

[1]Department of Pharmacy Practice, Saastra College of Pharmaceutical Education & Research, Jwalamukhi temple, Varigonda, Nellore, Andhra Pradesh 524311

[2]Saastra College of Pharmaceutical Education & Research, Jwalamukhi temple, Varigonda, Nellore, Andhra Pradesh 524311

Article History:

Received on: 25 Apr 2023
Revised on: 16 May 2023
Accepted on: 17 May 2023

Keywords:

Tertiary Care,
Prescription,
Diabetes Mellitus,
Hyperglycemia

ABSTRACT

Hyperglycemia produced on by type-1 or type-2 diabetes mellitus characterises the complex group of metabolic disorders known as diabetes mellitus. Because concomitant conditions can aggravate DM as well as increase the risk of complications, it is important to evaluate the prescription patterns in these individuals. The unique and general characteristics of diabetes patients, such as the available dose forms, the unexpected administration of medications to patients, and reported drug interactions, including common co-morbidities encountered in diabetic patients, all add to the challenges that the practitioner treating individuals. Due to the aforementioned factors, the study was created to help reduce prescription errors, provide safe dosage regimens, educate patients by closely monitoring the patients' glycaemic control and other responses to therapy, as well as promote the responsible and sensible use of medications. This study used questionnaires as a tool and was a prospective observational study that lasted six months. The study is being done at Global Hospital Lb. Nagar's medical ward. Those who visited OP and were admitted to the hospital's Medicine ward between October 2016 and March 2017 are included in the study. The following requirements must be met for a patient to enlist. Males (62.30%) and females (32.90%) made up the study's gender distribution. The age distribution was determined to be as follows: 30–35 years (8%), 35–45 years (14%), 45–55 years (56%), 55–65 years (38%), 65–75 years (19%), and 75–85 years (10%). The comorbid diseases identified in the 94 patients overall were hypertension, hyperthyroidism, chronic renal disease, infections of the urinary tract, as well as coronary artery disease (CAD).

*Corresponding Author

Name: Gautham Chakra R
Phone: +91 7674016126
Email: gauthamrowdhra05@gmail.com

eISSN: 2583-116X pISSN:

DOI: https://doi.org/10.26452/ijebr.v2i2.514

Production and Hosted by
Pharmasprings.com

INTRODUCTION

Hyperglycemia brought on by type-1 or type-2 diabetes mellitus characterises the complex group of metabolic illnesses known as diabetes mellitus.

More than 346 million individuals globally, according to the WHO, develop DM (1). Without any help, this number is more likely to quadruple by 2030 [1].

Diabetes is a difficult condition to adequately treat. Because diabetic nephropathy is a significant contributor to early morbidity, this study aims to evaluate the function of clinical chemists in addressing it. 40% of all patients in the United Kingdom who require dialysis have kidney failure as a result of

diabetic nephropathy, which is a significant source of early morbidity and mortality in individuals with diabetes mellitus (I). Hyperglycemia brought on by type-1 or type-2 diabetes mellitus characterises the complex group of metabolic illnesses known as diabetes mellitus.

More than 346 million individuals globally, according to the WHO, develop DM (1). Without any help, this number is more likely to quadruple by 2030 [2].

Diabetes is a difficult condition to adequately treat. Because diabetic nephropathy is a significant contributor to early morbidity, this study aims to evaluate the function of clinical chemists in addressing it. 40% of all patients in the United Kingdom who require dialysis have kidney failure as a result of diabetic nephropathy, which is a significant source of early morbidity and mortality in individuals with diabetes mellitus (I). Approximately 10% as well as 30%, respectively, of people with type 1 (insulin-dependent) diabetes mellitus (IDDM) develop nephropathy within 10 and 20 years, respectively. Nearly 20% of diabetics under the age of 50 die from nephropathy, which is also strongly linked to an elevated risk of cardiovascular disease. Additionally, it takes less time to develop in people with type I1 (non-insulin-dependent) diabetes mellitus (NIDDM). Antihypertensive medication has long been recognised for its advantages in reducing the rate of deterioration in renal function, which has made the need for effective therapeutic intervention to postpone or prevent renal failure development a priority. Angiotensin converting enzyme (ACE) inhibitors, however, have recently produced some quite impressive results. Whether ACE inhibitors can stop the progression of early disease with micro-albuminuria to advanced nephropathy is still unknown [3].

Classification of Diabetes

The majority of diabetic patients split into one of two broad categories: type 1 diabetes, which is brought on by a complete lack of insulin, or type 2 diabetes, which is characterised by the presence of insulin resistance and an insufficient increase in insulin secretion as a form of compensatory insulin resistance [4, 5].

Type 1 diabetes:

The pancreatic cells are destroyed autoimmunely in this type of diabetes. 90% of people have antibodies against insulin, glutamic acid decarboxylase, and islet cells at the time of diagnosis, which are markers of immunological destruction of the cell. Although this type of diabetes typically affects kids and teenagers, it can affect people of any age. While adults often experience LADA for many years, younger people often have a high rate of -cell breakdown as well as manifest with ketoacidosis.

Type 2 diabetes:

This form of diabetes is characterized by insulin resistance and a relative lack of insulin secretion, with progressively lower insulin secretion over time. Most individuals with type 2 diabetes exhibit abdominal obesity, which causes insulin resistance. In addition, hypertension, dyslipidaemia (high triglyceride levels and low HDL-cholesterol levels), and elevated plasminogen activator inhibitor type 1 (PAI-1) levels are often present in these individuals. Type 2 diabetes has a strong genetic predisposition and is more common in all ethnic groups other than those of European ancestry.

Epidemiology:

In 2011, 366 million people were expected to have DM; by 2030, this number will have increased to 552 million. Every country is seeing an increase in the prevalence of type 2 diabetes, with 80% of those affected residing in low- and middle-income nations. In 2011, 4.6 million people perished from DM. By 2030, 439 million individuals are predicted to develop type 2 diabetes. Due to environmental as well as behavioural risk factors, there are significant regional differences in the incidence of type 2 diabetes. In the next 20 years, it is expected that the prevalence of diabetes in adults in which type 2 DM is becoming more prevalent—will rise. A large portion of this rise will take place in emerging nations, where the majority of patients are between the ages of 45 as well as 64 [6].

Etiology:

When the pancreas stops producing enough insulin or when the body becomes resistant to insulin, type 2 diabetes can occur. Although the exact cause of this is uncertain, environmental as well genetic variables, including obesity as well as inactivity, appear to play a role [7].

Signs and Symptoms:

Type 2 diabetes can be present for years without symptoms. Keep an eye out for: increased hunger, increased thirst, as well as frequent urination. losing weight, Fatigue, vision fuzziness, slow-healing wounds or recurrent infections, darker skin patches [8].

Risk Factors:

Some factors include—such as body weight, distribution of fat, increased appetite, excessive thirst, weight loss, more frequent urination, blurred eye-

sight, excessive exhaustion, sores that don't heal, inactivity, ancestry, race, age, as well as Pregnancy-related diabetes, polycystic ovaries, as well as prediabetes [9].

Diabetic Emergencies:

People with type 1 as well as type 2 DM frequently have low blood sugar. The majority of instances are minor and are not emergencies. Effects might range from uneasy sensations, shaking, sweating, and increased appetite in mild cases to more serious problems like confusion, behavioural abnormalities, seizures, unconsciousness, and (rarely) fatal brain damage or death in serious instances. Self-medication for mild instances involves consuming sugar-rich foods as well as beverages. Serious cases, which can cause coma, need to be treated with intravenous glucose or glucagon infusions [10].

Treatment

Pharmacological therapy for type 2 DM:

Improved glucose control as well as fewer long-term problems are linked to early pharmacologic therapy beginning in type 2 diabetes. The following drug classes are utilised in the management of type 2 diabetes [11]:

1. Sulfonylureas
2. Meglitinide derivatives
3. Biguanides
4. Alpha-glucosidase inhibitors

Thiazolidinediones (TZDs) Insulin treatment was only given to type 2 diabetic patients whose blood sugar levels could not be controlled by oral medications and dietary adjustments. However, there is mounting evidence that early use of insulin may enhance overall diabetes management as well as the maintenance of the pancreas' capacity to produce insulin.

Some diabetic individuals only received insulin treatment if their type 2 diabetes could not be managed with oral medications as well as dietary adjustments. In contrast, there is mounting evidence that early use of insulin may enhance overall diabetes control as well as the maintenance of the pancreas's capacity to produce insulin.

Insulin therapy

]Some persons with type 2 diabetes also require insulin therapy. Because of its advantages, insulin therapy is now frequently administered earlier than it ever was. Insulin needs to be administered since it is hampered by regular digestion. Your doctor may advise using a variety of insulin kinds both during the day and at night, depending on your needs. People with type 2 diabetes frequently begin using insulin with one long-acting injection at night. A fine needle as well as syringe or an insulin pen injector, which resembles an ink pen but has an insulin cartridge instead of ink, are used to provide injections of insulin [12].

There are numerous varieties of insulin, as well as each one functions differently. Options consist of:

Insulin glulisine (Apidra)

Insulin lispro (Humalog)

Insulin aspart (Novolog)

Insulin glargine (Lantus)(38)

Insulin detemir (Levemir)

Insulin isophane (Humulin N, Novolin N)

Describe to your doctor the benefits and drawbacks of certain medications. After carefully weighing several considerations, such as expenses and other aspects of your health, you can select which drug is ideal for you. Your doctor may also recommend blood pressure and cholesterol-lowering drugs in addition to diabetes meds that will prevent heart and also the blood vessel disease, as well as low-dose aspirin therapy.

Bariatric surgery

If you have type 2 diabetes and a body mass index (BMI) above 35, you might be a candidate for bariatric surgery, which involves weight loss. Depending on the method used, blood sugar levels return to normal in 55 to 95 percent of diabetic patients. Other weight-loss procedures have less of an impact on blood sugar levels than surgeries that bypass a part of the small intestine [Figures 1, 2 and 3] [13].

Pre-mixed insulin, which is a combination of intermediate-acting and short- or rapid-acting insulin in a single bottle or insulin pen, may be an alternative in some circumstances [Figures 4, 5 and 6].

METHODOLOGY

MATERIALS AND METHODS

Source of data

Case report forms of type II Diabetes Mellitus patients.

Rationality of the Study

Table 1: Oral Agents for the Treatment of Type 2 DM

Class	Primary Mechanism of Action	Agent(s)	Available as
Sulfonylureas	The beta cells' initial impact is to secrete more insulin; they may also slow the rate at which the liver produces glucose as well as improve the sensitivity of the insulin receptor.	Acetohexamide Chlorpropamide Tolazamide Tolbutamide Glipizide Glyburide Glimepiride	Dymelor Diabinese Tolinase Orinase Glucotrol DiaBetaMicronase Amaryl
Short-acting insulin secretagogues	Interacting with K+ channels on beta islet cells increases insulin production. lowers post-meal hyperglycemia. Existing glucose levels determine how much insulin is released.	Nateglinide Repaglinide	Starlix Prandin
Thiazolidinediones	Elevates target-cell responses to insulin; lowers hepatic gluconeogenesis; action dependent on insulin.	Pioglitazone Rosiglitazone	Actos Avandia
Sodium-glucose cotransport-2 (SGLT-2) inhibitor	Sodium-glucose transporter-2 (SGLT2) inhibitor with high selectivity.	Dapagliflozin	Farxiga
Amylin analogue	Reduce the production of glucagon Gastric emptying gradually Enhance appetites	Pramlintide	Symlin
a-Glucosidase inhibitors	Delayed intestinal carbohydrate absorption	Acarbose Miglitol	Precose or generic Glyset
Glucagon-like peptide-1 (GLP-1) receptor agonists (Injectable drugs)	A glucagon-like peptide-1 (GLP-1) that suppresses glucagon, slows stomach emptying, and acts as an incretin mimic.	Exenatide Liraglutide	Byetta Victoza
Biguanides	Reduce HGP Increase in muscle glucose absorption	Metformin	Glucophage or generic
Bile acid sequestrant	Reduce HGP Elevating incretin levels	Colesevelam	WelChol
DPP-4 inhibitors	Boost the release of glucose-dependent insulin Reduce the production of glucagon	Alogliptin Linagliptin Saxagliptin Sitagliptin	Nesina Tradjenta Onglyza Januvia
Dopamine-2 agonist	Makes dopaminergic receptors active	Bromocriptine	Cycloset, Parlodel

Table 2: Insulin Therapy for Type 2 DM

Insulin type	Generic and brand names	Onset	Peak	Duration
Rapid-acting	Insulin aspart (NovoLog) Insulin glulisine (Apidra) Insulin lispro (Humalog)	15 min	30 to 90 min	3 to 5 hours
Short-acting	Insulin regular (HumulinR, Novolin R)	30 to 60 min	2 to 4 hours	5 to 8 hours
Intermediate-acting	Insulin NPH human (HumulinR,Novolin N)	1 to 3 hours	8 hours	12 to 16 hours
Long-acting	Insulin glargine (Lantus) Insulin detemir	1 hour	No clear peak	20 to 26 hours

Table 3: Various drugs prescribed for diabetic patients with hypertension, ischemic heart disease, and dyslipidaemia

Comorbidity	Drugs prescribed	Total drug usage (%)	Controlled diabetic patients (%)	Uncontrolled diabetic patients (%)
Hypertension	CCBs	21.44	29.0	16
	β-Blockers	14.30	14.37	14.24
	AT1-antagonists	19.40	14.3	22.72
	ACE inhibitors	2.05	4.12	2.6
	α-Antagonist	2.05	4.12	2.6
	Combinations	8.17	9.25	7.4
IHD	Clopidogrel	12.2	9.4	12.55
	Aspirin	3.0	6.6	2.6
	Combinations	4.0	4.12	4.084
Dyslipidaemia	Statins	19.3	22.07	17.63

Table 4: Drug Prescribing Pattern

Drug prescribing pattern		
Items	Drugs	%
Drug Groups	Antidiabetic	22%
	Antihypertensives	15.5%
	Multivitamins	12.72%
	Antiplatelet	9.9%
	Statins	4.31%
	Miscellaneous category	35.5%
Antidiabetic drugs	Metformin	43.14%
	Glimepiride	8.83%
	Vildagliptin	0.98%
	Sitagliptin	1.96%
	Insulin	42.16%
Fixed Dose Combinations (FDCs)	Metformin + Glimepiride	3.9%

Table 5: Demographic Details of Study Population

S.No	IP.No	Age	Sex	Reasons For Admission
1	23751	76	M	low back pain, fever for 10 days, generalized body weakness
2	28186	57	F	fever, redness 15 days, c/ o diffuse swelling in the left foot.
3	27467	64	M	sudden onset of weakness in left UL and LL
4	26840	35	M	Giddiness & profuse sweating
5	25420	60	M	Acute abdominal pain, constipation, nausea & vomiting, SOB
6	28001	54	F	Fever with chills & rigours
7	28002	47	F	Fever with cold & cough with sputum, abdominal pain loose stools
8	27751	78	F	Giddiness increased with a change in head position,
9	27791	34	M	Generalised weakness, difficulty in climbing up stairs
10	28195	40	F	High-grade fever with chills, headache, vomiting pains
11	28083	43	M	Fever associated with headache, generalised body pains
12	26116	77	M	loss of speech with right UL weakness since 3D
13	28213	52	M	unconscious state associated with sweating, loose stools, and fever for 1 day.
14	28128	36	M	fever on & off associated with expectoration, black stools.
15	28109	76	M	vomiting, slurred, slurred speech, generalised weakness, and chest pain.
16	28761	68	M	pain in the abdomen, previously had seizures
17	27573	48	F	chest pain, SOB associated with sweating, pain at left radiating.
18	27333	55	M	Sudden onset of giddiness, fall at home, fever.
19	26180	57	F	Abdominal pain at right hypochondria fever
20	27765	79	F	left UL & LL weakness, loose stools.
21	27346	58	M	right-sided weakness in both UL & LL associated with mouth deviation
22	28275	65	M	jaundice, fever, coloured urine
23	22491	55	M	retrosternal & epigastric burning associated with sweating
24	27922	62	F	generalizes weakness & history of reduced sleep, and joint pains.
25	28496	50	M	hoarseness in voice, walking at 3 a.m. to the toilet & unconscious
26	25680	42	F	abdominal distention, pain in abdomen
27	27577	47	M	Black-coloured stools, vomiting
28	21641	38	F	lower abdominal pain, chest discomfort, SOB, mild giddiness

Continued on next page

Table 5 continued

S.No	IP.No	Age	Sex	Reasons For Admission
29	28992	38	F	severe knee joint pain (right side), fever, loose stools
30	21660	64	M	Fever, cough with expectoration in the last 3-4 days
31	27798	75	M	giddiness associated with LOC
32	28640	64	M	weakness of left UL&LL, slurring of speech with a deviation of the mouth
33	23307	65	M	Fever with chills & rigours, body aches in the last 3 months
34	26781	53	F	pain in the left hip, bed sores grade 2 on the right gluteal region
35	26661	68	F	breathlessness on exertion, fatigue, dull, intermediate palpitation
36	28368	68	M	giddiness,1-episode seizure
37	28357	55	M	non-healing ulcer over left toe in the last 1 month
38	28625	64	M	multiple episodes of loose stools, cramps& abdominal pain
39	26722	55	M	sudden onset of left half-body paraesthesia decreased sensation
40	27572	78	F	slurring of speech, mouth deviated to the right side
41	27368	50	F	sudden giddiness& chest discomfort
42	28188	38	M	F/U/C of post left URSL+ DJS
43	28056	56	M	High-grade fever, bleeding manifestation like rashes & joint pains
44	26367	70	F	Low-grade fever, cough, loss of appetite
45	28013	36	M	nephrotic syndrome, old PTE, came for renal biopsy.
46	26655	70	F	fever, dry cough, and body pains since 10D
47	26667	34	M	Worsening, weakness, difficulty in sitting and walking, weight loss:10kgs
48	27602	58	F	uncontrolled sugar, leg pain, heaviness in chest.
49	27673	44	M	loose stools, pain in the abdomen, post renal transplant.
50	28043	36	M	came for a renal biopsy
51	28994	68	M	Fever, Left leg pain
52	29016	25	M	right radio cephalic fistula
53	28289	56	M	AV fistula constriction
54	28732	60	F	SOB, orthopnoea, chest discomfort
55	28955	58	F	Headache, generalised weakness, Dysarthria.
56	28676	56	M	Fell at his home and sustained injury over his Left face, Chest and Knee
57	26736	58	F	SOB, Vomiting, Typical Chest Pain.
58	28696	61	F	AV fistula constriction
59	28337	48	M	Pain and weakness in right ankle, painful flexion
60	28004	36	F	Left-sided chest pain radiating to Arm.
61	28843	66	M	SOB and Bilateral Pedal Edema.

Continued on next page

Table 5 continued

S.No	IP.No	Age	Sex	Reasons For Admission
62	28344	53	M	Hematemesis,Malena,SOB Grade II
63	28930	85	M	Weakness, unresponsiveness, sensorium.
64	28849	54	M	Abdominal Pain.
65	28056	56	M	High-Grade fever.
66	28274	55	F	High-grade fever with chills and rigours, Dry Cough.
67	27981	66	M	Unconscious
68	28991	70	M	Fever with chills, rigours, giddiness.
69	28936	68	F	SOB, vomiting, Epigastric discomfort.
70	28489	64	M	Fever, Burning Micturition admitted for cystoscopy.
71	28858	57	F	Chest discomfort, SOB, Orthopnoea.
72	28429	61	M	Pain in the right shoulder.
73	28857	56	M	Orthopnoea, chest discomfort.
74	28619	65	F	unresponsive state, sensorium
75	28956	58	M	headache. generalizes weakness, and dysarthria.
76	28675	49	M	admitted for AV fistula constriction
77	28799	59	M	complete body ache, abdominal discomfort, low back pain
78	28716	53	M	bilateral oedema, abdominal distention, scrotal swelling
79	27854	53	M	upper abdominal pain associated with vomiting, and fever.
80	27361	61	M	acute urinary retention, dysuria
81	26245	61	F	fever, cough with sputum, weakness, SOB on excretion.
82	27502	57	F	Syncope with sweating, palpitation
83	28064	67	M	Unresponsive state, not moving limbs.
84	26612	78	M	SOB on exertion, cough with sputum, oedema on both legs.
85	28083	38	M	Fever with chills, General body pains, Headache.
86	28635	54	F	Recurrent attacks of pyelonephritis
87	27989	61	F	Fever, chest pain, lower back pain, vomiting, Right cheek swelling.
88	27575	65	M	SOB.
89	27460	60	F	Fever, cough, weakness, headache, LOC.
90	27745	60	M	Fever, SOB, Sleep disturbance.
91	27705	53	M	pain in the abdomen, radiating to right loin, vomiting 2 episodes.
92	26604	71	M	cough with sputum, pain in the lower abdomen
93	26661	68	M	difficulty in breathing.low abdominal pain, dark stools
94	21733	52	M	fever, swelling of feet, generalised weakness, loss of appetite

Table 6: Demographic details of the study population Drug Interactions

IP No	Drug Interactions	DI Effects	Severity
23751	Azithromycin + Ondansetron	both increase Qt, CHF, bradycardia, and electrolyte imbalance	Serious
28186	Linezolid + Tramadol	both increase serotonin levels, monitor CNS & renal toxicity	Serious
27467	Labetalol + Amlodipine	both increase anti-HTN channel blocking	Significant
26840	Biseprolol+ Amlodipine/Biseprolol+ Kcl	anti-HTN blocking/increases Sr. K+	Significant
25420	Ofloxacin + Metformin	increases the effect of metformin by the PD effect.	Significant
28001	Pantoprazole + Clopidogrel	decreases the effect of clopidogrel by affecting the hepatic enzyme CYP2C19 metabolism	Significant
28002	NO	NO	No
27751	Losartan + Aspirin	increases Sr.K+ levels, renal function deterioration	Significant
27791	NO	NO	No
28195	Azithromycin + Ondansetron	both increase Qt, CHF, bradycardia, and electrolyte imbalance	Serious
28083	Escitalopram + Clopidogrel	risk of bleeding	Significant
26116	NO	NO	Minor
28213	NO	NO	No
28128	Clarithromycin + Amlodipine	increases the effect of Amlodipine enzyme CYP3A4 metabolism	Significant
28109	Cinnarizine + Prochlorperazine	increases sedation	Significant
28761	NO	NO	No
27573	Ceftriaxone + Enoxaparin	increases anticoagulant of enoxaparin	Serious
27333	Pantaprazole + Clopidogrel	decreases the effect of clopidogrel by affecting the hepatic enzyme CYP2C19 metabolism	Significant
26180	NO	NO	No
27765	Telmisartan + Atorvastatin	increases myopathy	Significant

Continued on next page

Table 6 continued

IP No	Drug Interactions	DI Effects	Severity
28275	Metronidazole + Acetaminophen	increases PD effect	Minor
22491	Heparin + Aspirin	increases anticoagulant	Significant
27922	Aspirin + Glimepiride	increases the effect of glucose by an unknown mechanism	Significant
27922	Aspirin + Glimepiride	increases the effect of glucose by an unknown mechanism	Significant
28496	NO	NO	No
25680	NO	NO	No
27577	Fluconazole + Ondansetron	increases QTC interval	Serious
21641	NO	NO	No
27798	Bisoprolol + Amlodipine	both increase anti-HTN channel blocking	Significant
28640	Telmisartan + Atorvastatin	increases toxicity of atorvastatin	Significant
23307	Hydrocortisone + Levofloxacin	both increase the synergism.	Significant
26781	NO	NO	No
26661	Levofloxacin + Metformin	increases the effect of metformin by PD synergism	Significant
28368	NO	NO	No
28357	Ofloxacin + Metformin	increases the effect of metformin by the PD effect.	Significant
28625	Pantoprazole + Clopidogrel	decrease the effect of clopidogrel by affecting the hepatic enzyme CYP2C19 metabolism	Significant
26722	Pantoprazole + Clopidogrel	decrease the effect of clopidogrel by affecting the hepatic enzyme CYP2C19 metabolism	Significant
27572	Losartan + Carvedilol	both increase serum potassium	Significant
27368	Pantoprazole + Clopidogrel	decrease the effect of clopidogrel by affecting the hepatic enzyme CYP2C19 metabolism	Significant
28188	NO	NO	No

Continued on next page

Table 6 continued

IP No	Drug Interactions	DI Effects	Severity
28056	Sodium Bicarbonate + Azithromycin	decreases the level of azithromycin by inhibition of GI absorption	Significant
26367	Pantoprazole + Dabigatran	increases the level of dabigatran by P-Glycol protein(MDR1)	Significant
28013	Aspirin + Furosemide	increases and decreases serum potassium	Significant
26655	Pantoprazole + Budesonide	decreases the effect of budesonide by increasing gastric Ph	Significant
28043	Metformin + Furosemide	decreases the level of Furosemide by an unspecified mechanism	Minor
28289	Clonidine + Metoprolol	increases the risk of bradycardia	Serious
28732	Telmisartan+Atorvastatin	increases the risk of myopathy	Significant
28676	Pantaprazole+Cyanocobalamine	decreases the level of cyanocobalamin by inhibiting GI absorption	Minor
26736	Na Bicarbonate+Levofloxacin	decreases the level of levofloxacin by inhibition of GI absorption	Serious
28696	No	NO	No
28337	Rantidine+Cyanocobalamine	decreases the level of cyanocobalamin by inhibiting GI absorption	Minor
28004	Olmisartan+Aspirin	increases serum potassium levels	Significant
28843	Aspirin+Prasugrel	increases the toxicity by PD synergism	Significant
28344	Octreotide+Ondansetron	both increase the QT interval	Serious
28930	Carvedilol+ Hydralazine	increases the effect of carvedilol by PD synergism	Significant
28849	Isoniazide+Ondansetron	CYP-450 inhibitor may decrease the clearance of ondansetron	Significant
28056	Azithromycin + Ondansetron	increases QTC interval	Serious

Continued on next page

Table 6 continued

IP No	Drug Interactions	DI Effects	Severity
28274	Pantaprazole+Cyanacobalamine	decreases the level of cyanocobalamin by inhibiting GI absorption	Minor
27981	Pantaprazole+Clopidogrel	decreases the effect by affecting the hepatic enzyme CYP2C19 metabolism	Significant
28991	No	NO	No
28936	Pantaprazole+Cyanacobalamine	decreases the level of cyanocobalamin by inhibiting GI absorption	Significant
28489	Piperacillin+Amikacin	increases the effect of amikacin by PD synergism	Minor
28858	Olmisartan+Telmisartan	increases serum potassium levels	Significant
28429	Metoprolol+Cyanocobalamine	decreases the level of cyanocobalamin by an unexpected mechanism	Minor
28857	No	NO	No
28619	Atorvastatin+Azithromycin	increases the level of azithromycin by P-Glycol protein(MDR1)	Significant
28956	Telmisartan+Atorvastatin	increases the risk of myopathy	Significant
28675	Biseprolol+ Amlodipine/Biseprolo+ Kcl	increases and decreases serum potassium	Significant
28799	No	NO	No
28716	Aspirin+Furosemide	increases and decreases serum potassium	Significant
27854	No	NO	No
27361	Diclofenac+Budesonide	increases toxicity by PD synergism	Significant
26245	Pantaprazole+Clopidogrel	decreases the effect by affecting the hepatic enzyme CYP2C19 metabolism	Significant
27502	Azelastine+Cinnerizine	increases sedation	Significant
28064	Pantaprazole+Cyanacobalamine	decreases the level of cyanocobalamin by inhibiting GI absorption	Significant

Continued on next page

Table 6 continued

IP No	Drug Interactions	DI Effects	Severity
26612	Pantaprazole+Clopidogrel	decreases the effect by affecting the hepatic enzyme CYP2C19 metabolism	Significant
28083	Atorvastatin+Azithromycin	increases the level of azithromycin by P-Glyco protein (MDR1)	Significant
28635	Metformin+Furosemide	decreases the level of furosemide by an unknown mechanism	Minor
27989	Pantaprazole+Clopidogrel	decreases the effect by affecting the hepatic enzyme CYP2C19 metabolism	Significant
27575	Telmisartan+Furosemide	increases and decreases serum potassium	Significant
27460	Azithromycin + Ondansetron	both increase the QTc interval	Serious
27745	Budesonide+Hydrocortisone	decreases the level of hydrocortisone by affecting CYP3A4 metabolism	Significant
27705	Pantaprazole+Clopidogrel	decreases the effect by affecting the hepatic enzyme CYP2C19 metabolism	Significant
26604	Ceftizoxime+Furosemide	increases the toxicity of furosemide by PD synergism	Minor
26661	Ramipril+Glimeperide	increases the effect of glimepiride by PD synergism	Significant
21733	Pantoprazole + Cyanocobalamine	decreases the level of cyanocobalamin by inhibiting GI absorption	Significant
22474	Levodopa+Amlodipne	increases the effect of amlodipine by PD synergism	Significant
26304	No	NO	No
27822	Telmisartan+Metoprolol	both decrease the serum potassium level	Significant

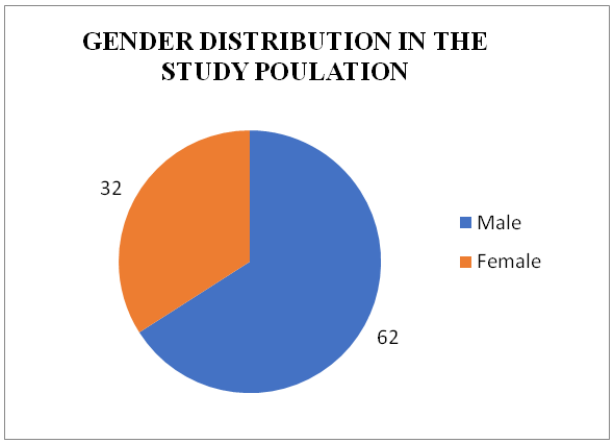

Figure 1: Based on Gender

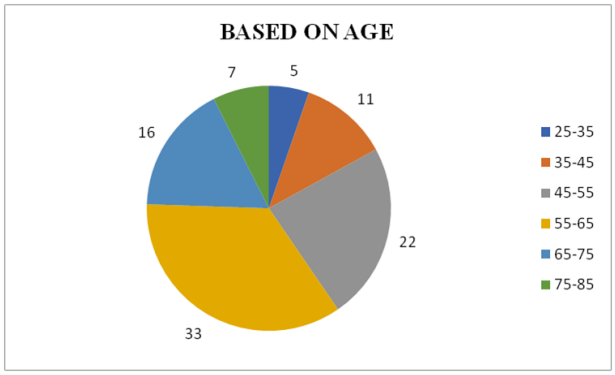

Figure 2: Based on Age

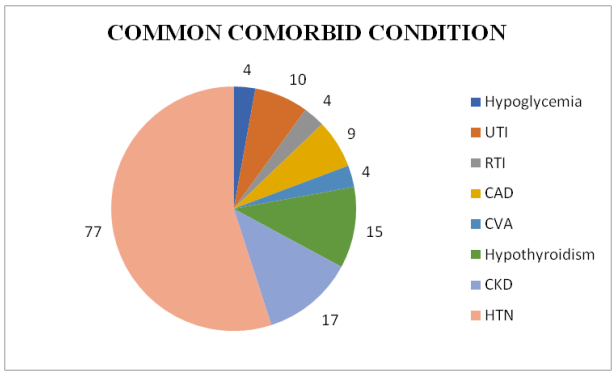

Figure 3: Based on Common Comorbidities

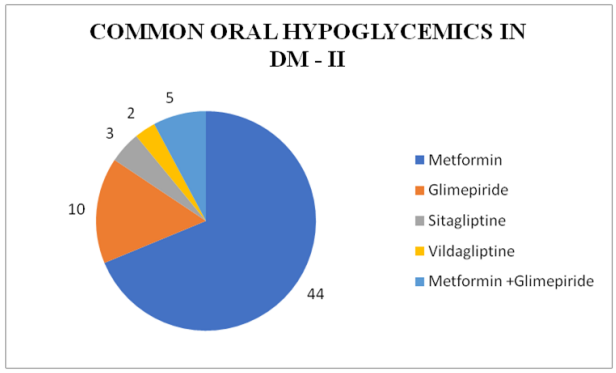

Figure 4: Based on common Oral Hypoglycemics

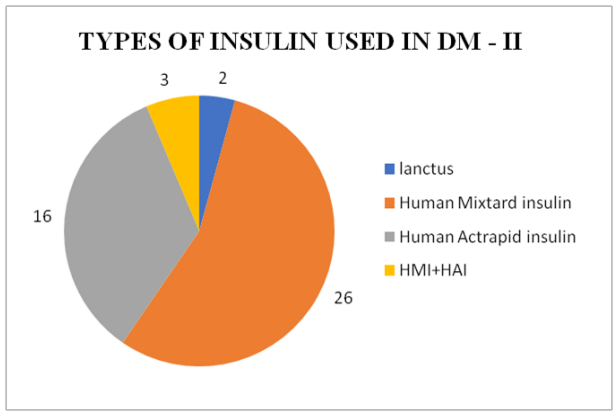

Figure 5: Based on types of Insulin used in Type 2 DM

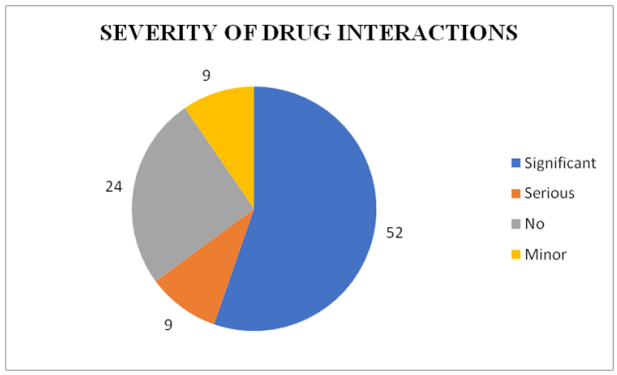

Figure 6: Based on Severity of Drug Interaction

The challenges a physician treating diabetes patients faces are exacerbated by both the unique and general characteristics of these patients, such as the dose forms that are available, the unanticipated administration of medications to patients, reported drug interactions, and typical co-morbidities encountered in diabetic patients. The study was created to aid in reducing prescription errors, providing safe dosage regimens, teaching the patients by closely monitoring the patients' glycaemic control and other responses to medication, and ultimately promoting the judicious and sensible use of pharmaceuticals [Tables 1 and 2].

Method and Collection of Data

Study site

The study conducted at Medicine ward of Gleneagles Aware Global Hospital LB. Nagar.

Study duration

The study was conducted for six months from October 2017 to March 2018 [14].

Study design

It is a prospective observational study conducted on diabetes mellitus patients.

Study criteria

Table 7: Based on Gender

Gender	No of patients	Percentage
Male	62	60.30%
Female	32	32.90%

Table 8: Based on Age

Age	No of patients	Percentage
25-35	5	5.00%
35-45	11	11.00%
45-55	22	23.00%
55-65	33	33.00%
65-75	16	16.00%
75-85	7	7.00%

Table 9: Based on Common Comorbidity

Common Comorbidity	No of Patients
Hypoglycemia	4
UTI	10
RTI	4
CAD	9
CVA	4
Hypothyroidism	15
CKD	17
HTN	77

Table 10: Based on Common Oral Hypoglycemics

Drugs	No of Patients
Metformin	44
Glimepiride	10
Sitagliptin	3
Vildagliptin	2
Metformin +Glimepiride	5

Table 11: Based on types of Insulin used in Type 2 DM

Insulin	No of Patients
Sanctus	2
Human Mixtard insulin	26
Human Actrapid insulin	16
HMI+HAI	3

Table 12: Based on Severity of Drug Interaction

Severity	No of Cases
Significant	52
Serious	9
No	24
Minor	9

The following study is carried out using the following factors,

Inclusion criteria

1. Patients with diabetes for at least 1 year.
2. Patients with diabetic complications.
3. Patients with other co-morbid conditions [15, 16].

Exclusion criteria

Pregnant women and nursing mothers.

Study Procedure

This is a prospective observational study conducted over six months. The study was conducted at Medicine ward of Gleneagles Aware Global Hospital LB. Nagar. Patients who were admitted to the Medicine ward of the hospital and those visiting OPD for six months from October 2017 to March 2018 are enrolled. Diabetic patients visiting the endocrinologist are evaluated, diagnosed, and prescribed suitable therapy. Using a suitable data collection form, the following details collected are patient demographics, prescription charts, lab data, progress charts, medical records, doctor's notes, and nursing notes [17].

RESULTS

This investigation was carried out in a tertiary care hospital's IPDs for diabetesology as well as general medicine. Participants in this study had to have type 2 diabetes for at least a year and be between the ages of 25 and 85, regardless of their gender. Patients older than 85 years old were eliminated due to the increasing occurrence of other co-existing illness conditions. Data were gathered from 98 diabetic patients' profile sheets who visited the OPD and IPD over the study's six-month period, from October 2017 to March 2018 [Tables 3 and 4].

Records are used to gather information on patient demographics, blood glucose and HbA1C levels, diagnoses, and medications administered [Tables 5 and 6].

Patients were categorised as having controlled fasting blood sugar (FBS) 110 mg/dL/HbA1C 7 or having uncontrolled diabetes (FBS >110 mg/dL/HbA1C >7) based on the blood glucose levels and HbA1C. The prescription patterns for medicines in managed and uncontrolled diabetics with additional comorbid illnesses were discovered using a descriptive analysis of the data. 62 (64.3%) of the 94 patients were men, and 32 (35.7%) were women, with respective mean ages of 58.06 11.13 as well as 57.08 12.58 years. 37 patients in our study population had diabetes that was under control, while 57 patients were not [Tables 7 and 8].

In the population that was under control, the meantime that type 2 diabetes persisted was 5.57 2.98 years, but in the uncontrolled group, it was 7.18 5.8 years [Tables 9 and 10].

The most prevalent cardiovascular comorbidity among diabetes patients was systemic hypertension, with a frequency of 78.6%. Among these individuals, 22% had IHD as well as 4% had dyslipidemia concomitant.IHD (49%) and dyslipidaemia (21%) came after systemic hypertension. In patients, 18.35% have hypothyroidism and 20% have CKD [Tables 11 and 12].

Additionally, aspirin was given to 4.1% of IHD patients, while 5.1% of patients also received clopidogrel. Statins were prescribed for all dyslipidemic individuals.

The patients with managed diabetes received higher CCB prescriptions. More individuals with uncontrolled diabetes than those with managed diabetes used combination antihypertensive medications. Only patients with uncontrolled diabetes received prescriptions for AT1 receptor blockers. individuals with uncontrolled diabetes received more prescriptions for clopidogrel, whereas individuals with managed diabetes received more prescriptions for aspirin.

The mean number of cardiovascular medications was found to be 1.12 0.58 among diabetics who were under control, compared to 1.52 1.10 in those who were not. The greater rate of uncontrolled diabetic patients may be a result of patients' poor adherence to therapy, lack of knowledge, and education. This could result in the need for extra medications or drug combinations to manage them.

Comorbid Conditions

Throughout the trial, 450 different medications were prescribed. Prescriptions for 101 (22%) anti-diabetics, 70 (15.5%) anti-hypertensives, 55 (12.72%) multivitamins, 44 (9.9%) anti-platelets, 18 (4.31%) statins, and 162 (35.5%) other medications were written. 42 (43.14%) of the patients receiving antidiabetics were administered metformin, 8 (8.83%) were prescribed glimepiride, 2 (1.96%) were prescribed sitagliptin, 1 (0.98%) were prescribed vildagliptin, and 41 (42.16%) were prescribed insulin. The most frequently administered FDC (4, 3.9%) was metformin plus glimepride.

DISCUSSION

Males (62.30%) and females (32.90%), respectively, made up the study's gender and age distributions. The age ranges were 25–35 years (5%), 35–45 years (11%) 45–55 years (23%) 55–65 years (33%) 65–75 years (16%), and 75–85 years (7%). The comorbid illnesses identified in the 94 instances in total were hypertension, hypothyroidism, chronic renal disease, urinary tract infections, and coronary artery disease.

Throughout the trial, 450 different medications were prescribed. Prescriptions for 101 (22%) anti-diabetics, 70 (15.5%) anti-hypertensives, 55 (12.72%) multivitamins, 44 (9.9%) anti-platelets, 18 (4.31%) statins, and 162 (35.5%) other medications were written.

42 (43.14%) of the patients receiving antidiabetics were administered metformin, 8 (8.83%) were prescribed glimepiride, 2 (1.96%) were prescribed sitagliptin, 1 (0.98%) were prescribed vildagliptin, and 41 (42.16%) were prescribed insulin. The most frequently administered FDC (4, 3.9%) was metformin plus glimepride. In addition, aspirin was given to 4.1% of IHD patients, while 5.1% of patients received both clopidogrel and aspirin. Statins were given to every patient with dyslipidemia. Individuals with uncontrolled diabetes were more likely to be administered clopidogrel, whereas individuals with managed diabetes were more likely to be prescribed aspirin.

In patients with managed diabetes, CCBs were more frequently prescribed. those with uncontrolled diabetes used combination antihypertensive medications more frequently than those with managed diabetes. Only those patients with uncontrolled diabetes received prescriptions for AT1 receptor blockers. The greater proportion of uncontrolled diabetes patients may be a result of their poor adherence to therapy, lack of knowledge, and education. To treat their comorbid diseases, extra medications or drug combinations can be required as a result.

The drug interactions that were discovered were categorised according to their severity as follows: 24 study population patients did not experience any drug interactions, 52 significant interactions, 9 serious interactions, and 9 mild interactions.

Standard treatment, which demonstrated effective control of the disease Diabetes + Hypertension, was as follows:

1. β-Blockers (15.31%) e.g. Metoprolol
2. Ca^{+2} channel blockers (22.45% usage) e.g. Amlodipine
3. Biguanides (43%) e.g. Metformin
4. Human Mixtard Insulin (25%)

CONCLUSION

In the final report, which was compiled from 94 patients with diabetes mellitus type II as the primary illness, hypertension was identified as the most common co-morbid condition (74 instances).

ACKNOWLEDGEMENT

The corresponding author desires to express gratitude to Dr. G. Rajeswari (Principal), Department of Pharmacology, Saastra College of Pharmaceutical Education & Research, Jwalamukhi temple, Varigonda, Nellore, India for her guidance and constant support in completing this research work.

Conflict of Interest

The author declares there is no conflict of interest.

Funding Support

The author declares there is no funding support.

REFERENCES

[1] Bastaki SS. Diabetes mellitus and its treatment. International Journal of Diabetes and Metabolism. 2005;13(3):111–134.

[2] Williamson R. Diabetes Mellitus and its Treatment. BMJ. 1898;2:120–120.

[3] Kaveeshwar S. The current state of diabetes mellitus in India. Australasian Medical Journal. 2014;7(1):45–48.

[4] Virtanen S, Knip M. Type 1 diabetes-origins and epidemiology. The Lancet Diabetes & Endocrinology. 2020;8(5):368–369.

[5] Van Belle T, Coppieters K, Herrath V. Type 1 Diabetes: Etiology, Immunology, and Therapeutic Strategies. Physiological Reviews. 2011; 91(1):79–118.

[6] Volkov V, Rudenko E, Rokkina S, et al. On pathogenesis of arterial hypertension in type 2 diabetes mellitus. Diabetes mellitus. 2011; 14(2):53–55.

[7] Haffner S. Epidemiology of Type 2 Diabetes: Risk Factors. Diabetes Care. 1998; 21(Supplement_3):3–6.

[8] Misra P, Upadhyay R, Misra A, et al. A review of the epidemiology of diabetes in rural India. Diabetes Research and Clinical Practice. 2011; 92(3):303–311.

[9] Erratum. Diabetes Mellitus in Rural India. Epidemiology. 2011;22(1):134–134.

[10] Suh D. Disease Management Program For Diabetes Mellitus. The Endocrinologist. 1999; 9(5):379–388.

[11] Adeghate E, Schattner P, Dunn E. An Update on the Etiology and Epidemiology of Diabetes Mellitus. Annals of the New York Academy of Sciences. 2006;1084(1):1–29.

[12] Uloko A, Musa B, Ramalan M, et al. Prevalence and Risk Factors for Diabetes Mellitus in Nigeria: A Systematic Review and Meta-Analysis. Diabetes Therapy. 2018;9(3):1307–1316.

[13] Smith S. Multiple Risk Factors for Cardiovascular Disease and Diabetes Mellitus. The American Journal of Medicine. 2007;120(3):3–11.

[14] Yan J, Gushulak K, Van Aarsen CM, et al. Risk factors for recurrent emergency department visits for hyperglycemia in patients with diabetes mellitus. International Journal of Emergency Medicine. 2017;10(1).

[15] Mooradian A, Bernbaum M, Albert S. Narrative Review: A Rational Approach to Starting Insulin Therapy. Annals of Internal Medicine. 2006;145(2):125–125.

[16] Hirsch I, Bergenstal R, Parkin C, et al. A Real-World Approach to Insulin Therapy in Primary Care Practice. Clinical Diabetes. 2005; 23(2):78–86.

[17] Bradley C. Intensive insulin therapy in critically ill patients. Intensive and Critical Care Nursing. 2002;18:128–129.

Cite this article: Gautham Chakra R, Rajeswari G, Alekya B, Mahaboob Ali Sk, Noorul Kareem M, Prekshasri S. A Tertiary Care Hospital's Prescription Pattern for Diabetes Mellitus. Int. J.Exp. Biomed. Res. 2023; 2(2): 62-79.