Impact of Clinical Pharmacist Education on Clinical Outcome of Diabetic Patients

A Thesis submitted for fulfillment of Master Degree in Pharmaceutical Sciences
(Clinical pharmacy)

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List of contents

List of tables

List of figures

List of abbreviations

ACCP	**AmericanCollegeClinicalPharmacy**
ADA	**AmericanDiabetesAssociation**
ASHSP	**AmericanSocietyHealthSystemPharmacy**
ANOVA	**Analysis Of Variance**
CPG	**ClinicalPracticeGuideline**
DCCTRG	**DiabetesControlComplicationsTrialResearch Group**
DM	**Diabetes Mellitus**
FPG	**Fasting Plasma Glucose**
HbA_{1C}	**Glycated Haemoglobin**
IDDM	**Insulin-Dependent Diabetes Mellitus**
KAP	**Knowledge Attitude & Practice**
MHRs	**Maintenance Of Managed Health Records**
MODY	**Maturity-Onset Diabetes Of The Young**
MTM	**Medication Therapy Management**
NIDDM	**Non-Insulin-Dependent Diabetes Mellitus**
NPH	**Neutral Protamine Hagedon**
OGTT	**Oral Glucose Tolerance Test**
PBKPAQ	**Patients basic knowledge& practice assessment Questionnaire**

Abstract

Aim of the work:

To assess impact of pharmacist-initiated diabetic education on patients' knowledge and skills about diabetes, patients' glycemic control and reduces adverse events and frequency of acute complications.

Patients &Methods:

Prospective, randomized, controlled study. Patients presenting to Medicine and Endocrinology clinic, Ain Shams University, Cairo, were assessed for eligibility. Inclusion criteria included; diabetics on insulin, aged ≥ 17 years. Exclusion criteria included; ESRD, pregnancy, patients on another diabetic education programs. Patients were randomly assigned to; Control (non-pharmacist group); 30 diabetics subjected to regular care, or Test (pharmacist group); 30 diabetics subjected to pharmacist-initiated education. For both groups; demographics, patient basic knowledge and practice assessment questionnaire (PBKPAQ) and HbA1C were assessed initially and patients given an adverse events andfrequency incidence of acute complicationsself-reporting card. Test group was subjected to a systematic diabetic education. Follow up was done continuously for 3 months for both groups and final evaluation included; questionnaire assessment, adverse eventsandfrequency incidence of acute complications reporting and HbA1C levels.

Results:

Groups were comparable at baseline. After 3 months, test group's HbA1C levels significantly decreased ($p< 0.001$) as compared to their baseline levels, control group's levels increased with time. The PBKAQ was significantly ($p< 0.001$) increased in both groups, yet the increase was higher in the test group.

Test group showed a significant improvement ($p< 0.001$) in the individual items of PBKPAQ versus the control group. Test group had a significantly lower frequency of hypoglycemic ($p= 0.019$) and hyperglycemic episodes ($p= 0.02$) versus control group.

Conclusion:

Pharmacist initiated diabetic education improvedpatients' knowledge and practice about diabetes,patients' glycemic controlandreduces frequency of acute complications.

Keywords: Diabetic education, pharmacist, glycemic control

DIABETES MELLITUS

A. Definition:

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both.

The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels**(ADA, 2013a).**

B. Prevalence of diabetes:

No country is free of diabetes. The prevalence of diabetes is growing rapidly around the world and is a major cause of morbidity and mortality**(Guariguata et al., 2011).**

It has been estimated that the world prevalence of diabetes among adults aged 20–79 years, is expected to reach 6.4%, affecting 285 million adults, in 2010, and will increase to 7.7% and 439 million adults by 2030**(King et al., 2012).**

Diabetes has been previously estimated to be the sixth most important cause of disability burden in Egypt**(Taha et al., 2013)**, and it has been estimated that by the year 2030, Egypt will have at least 8.6 million adults with diabetes. Little data is available on the epidemiology of diabetes in Egypt**(Arafa and Amin, 2010)**.

Moreover, the prior study concluded that the crude prevalence rate of known diabetes in Egypt in 2008 was 4.07%. This prevalence increased with age, and especially higher age. The study recommended that there should be a public health policy that should educate the public on the risk factors for diabetes, and should implement guidelines for adequate control of this disease**(Arafa and Amin, 2010).**

C. Etiologic classification of diabetes mellitus: (ADA, 2013a)

1. **Type 1 diabetes:** which is to β-cell destruction, usually leading to absolute insulin deficiency and which can be: immune mediated or idiopathic.

2. **Type 2 diabetes:** that may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance.

3. **Gestational diabetes mellitus**

4. **Other specific types and etiologic factors:** Diabetes may be caused due to other disorders, diseases or etiological agents:

A. Genetic defects of β-cell function including:**(ADA, 2014)**

1. Chromosome 12, HNF-1α (MODY3)
2. Chromosome 7, glucokinase (MODY2)
3. Chromosome 20, HNF-4α (MODY1)
4. Chromosome 13, insulin promoter factor-1 (IPF-1; MODY4)
5. Chromosome 17, HNF-1β (MODY5)
6. Chromosome 2, *NeuroD1* (MODY6)
7. Mitochondrial DNA
8. Others.

B. Genetic defects in insulin action including:**(Lann and LeRoith, 2007**)

1. Type A insulin resistance
2. Leprechaunism
3. Rabson-Mendenhall syndrome

4. Lipoatrophic diabetes

5. Others.

C. Diseases of the exocrine pancreas including; Pancreatitis, Trauma/pancreatectomy, Neoplasia, Cystic fibrosis, Hemochromatosis, Fibrocalculouspancreatopathy and others**(Virmani, 2006)**.

D. Endocrinopathies including: Acromegaly, Cushing's syndrome, Glucagonoma, Pheochromocytoma, Hyperthyroidism, Somatostatinoma, Aldosteronoma and Others**(Koike et al., 2003)**.

E. Drug or chemical induced-diabetes including: Vacor, Pentamidine, Nicotinic acid, Glucocorticoids, Thyroid hormone, Diazoxide, β-adrenergic agonists, Thiazides, Dilantin, γ-Interferon, Others.

F. Infections induced diabetes including: Congenital rubella, Cytomegalovirus and Others.

G. Uncommon forms of immune-mediated diabetes:

1. "Stiff-man" syndrome

2. Anti-insulin receptor antibodies

3. Others.

H. Other genetic syndromes sometimes associated with diabetes; Down syndrome, Klinefelter syndrome, Turner syndrome, Wolfram syndrome, Friedreich ataxia, Huntington chorea, Laurence-Moon-Biedlsyndrome, Myotonic dystrophy, Porphyria, Prader-Willi syndrome and Others**(ADA, 2013a).**

1. Type 1 Diabetes Mellitus

Type 1 DM was previously called insulin-dependent DM (IDDM) or juvenile-onset diabetes**(Narendran et al., 2005)**.

Type 1diabetes is thought to be inherited in genetically susceptible individuals by environmental factors such as viral, toxic or chemical agents that lead to autoimmune destruction of β-cells, resulting in the formation of altered protein components. This material is a foreign antigen to the immune system, establishing the basis for an auto-immune reaction against the cell of origin the β–cell**(Bosi et al., 1987; Betterle et al., 1994)**, which are the only cells in the body that synthesize insulin hormone that regulates blood glucose level. Insulin hormone "unlocks" the cells of the body, allowing glucose to enter and fuel them. This form of diabetes usually strikes children and young adults, although disease onset can occur at any age.

Insulin –dependent DM can either be immune –mediated or idiopathic. Type1may account for 5% to 10% of all diagnosed cases of diabetes**(Narendran et al., 2005)**. About 40% of people with type 1 develop severe nephropathy and kidney failure by the age of50**(Epstein et al., 1994)**.

2. Type 2 Diabetes Mellitus

Type 2diabetes was previously called non-insulin-dependent diabetes mellitus (NIDDM) or adult-onset diabetes. It may account for about 90% to 95% of all diagnosed cases of diabetes, it usually begins as insulin resistance**(Vaag, 2008)**.

Two groups of patients with type2 were categorized according to the different body composition, obese and non-obese. In addition, a third group called maturity-onset diabetes of the young (MODY) had been

described for those individuals in whom the diagnosis of diabetes is made before the age of 25 years**(Fajans, 1989)**.

Type 2 diabetes is usually associated with a positive family history, and begins in Middle life or beyond, often over the age of 40. Symptoms being more gradually than in type1, and the diagnosis is frequently discovered when an asymptomatic person is found to have elevated plasma glucose on routine laboratory examination. In contrast to type1, plasma insulin levels are normal to high due to the inability of insulin to lower plasma glucose levels effectively; an-abnormality termed insulin resistance**(ADA, 2004a; Attalah, 2007)**.

Type 2 diabetes can result from genetic defects that cause both insulin deficiency and insulin resistance and occurs during the early phase of type2, but the disease frequently goes undiagnosed for many years because hyperglycemia during the earlier stages is not severe enough to cause symptoms**(ADA, 2004a; Attalah, 2007)**.

The pathophysiologic alterations in type 2diabetes include abnormal insulin secretion and resistance to insulin action in target tissues. Although either defects may be the initial pathogenic event that ultimately leads to the disease, most patients with the fully developed syndrome show impairments of bothinsulin secretion and insulin-mediated glucose disposal (insulin or insulinantibodies), receptors (decreased number or diminished binding of insulin) or post receptor (Abnormal signal transduction especially failure to activate tyrosine kinase) abnormalities. Obesity is the most common cause of insulin resistance**(DeFronzo et al., 1992; Attalah, 2007).**

A comparison between type 1 and type 2 DM is represented in table 1.

Table (1):Comparison between type 1 & 2 DM(DeFronzo et al., 1992; Attalah, 2007)

	Type 1	**Type 2**
Age at onset	Usually under 40	Usually over 40
Body weight	Thin	Usually overweight
Symptoms	Appear suddenly	Appear slowly
Insulinproduced	None	Too little, or is ineffective
Insulinrequirement	Must take insulin	May require insulin (20-30%)
Symptoms	Usually abrupt; thirst,polyuria, polyphagia,weight loss	Frequently asymptomatic orthirst, fatigue visual blurringand easy fatigability
Nutritionalstatus	Usually non-obese	60-80% obese
Etiology	Viral or otherenvironmental factors	Family history & strong association with obesity.
Pathogenesis	Chronic autoimmunityagainst islet β-cells; islet cell antibodies detected years before clinical onset	Impaired insulin secretion and/or insulin resistance
Diet	Mandatory	Mandatory: diet alone may control blood sugar.
Islet cell antibodies at diagnosis	yes	No

3.Gestational diabetes:

Gestational diabetes mellitus is defined as glucose intolerance or diabetes mellitus diagnosed for the first time during pregnancy. It results from a pregnant inability to mount sufficient insulin secretion to compensate for the increased nutritional needs of gestation. This is the result of an increased adiposity of pregnancy and the anti–insulin hormones specifically; human placental lactogen, prolactin, cortisol and progesterone. In normal pregnancy, the insulin secretory response increases up to 4-fold to compensate for the diabetic forces of pregnancy**(Griffin et al., 2000).**

Gestational diabetes affects about 4% of all pregnant women, after pregnancy 5% to 10% of women with gestational diabetes develop type 2 diabetes**(Ben-Haroush et al., 2004**).

D. Diabetes Symptomatology:

Diabetes is sometimes discovered accidently in people who have no symptoms. Some people with diabetes have a variety of symptoms, which include**(Harris et al., 2003)**:

- Excessive thirst
- Frequent urination
- Dry skin
- Weight loss
- Frequent feeling of tiredness
- Slow healing of infections
- Blurred vision
- Tingling in the hands or feet

E. Diagnosis of Diabetes Mellitus:

The body usually is able to keep glucose concentrations stable. The normal fasting blood sugar is usually between70-120mg/dl (3.5-6.7mmol/l). After a meal it would rarely exceed 140 mg/dl (8mmol/l). Normally there is no glucose in urine since the normal threshold above which glucose would appear in the urine would be 10mmol/l. Below a concentration of 18 mg/dl 0(10mmol/l) the kidneys reabsorb glucose back into the blood stream and so glucose does not appear in the urine unless the blood concentration of glucose is high**(DCCTRG, 1993)**.