Republic of Iraq Ministry of higher Education and Scientific Research Qadisiyah University Faculty of Pharmacy



A field study compared the cases of high and low blood sugar therapies in children's hospitals in Diwanyah

A project

Submitted to the Qadisiyah university/Faculty of Pharmacy in partial of the requirement of Bachelors Degree in pharmaceutical science

> By: Noor Gassan Abd-Alhadi Yoser Ihsan Jabar

Supervised by: Lecturer Farah Razzaq Kbyeh

2017 AD

1

- وزارة التعليم العالي والبحث العلمي جامعة القادسية/كلية الصيدلة
 - فرع العلوم والمختبرات السريرية



دراسة ميدانية قارنت حالات ارتفاع وانخفاض نسبة السكر في الدم في مستشفيات الأطفال في الديوانية

الى كلية الصيدلة بجامعة القادسية كجزء من متطلبات نيل درجة بكالوريوس في الصيدلة

بحث مقدم

- مقدم من قبل نور غسان عبدالمهادي يسر احسان جبار
 - بأشراف م. فرح رزاق کبیح

2017



Acknowledgement

Praise be to god who enables our with his blessing to achieve this modest scientific effort.

We would like to express our deepest gratitude and appreciation to our supervisor *Lecturer Farah Razzaq Kbyeh* for suggesting the project, continuous advice and her guidance through the hard time of my work and we would like to thanks the *head of Collage of pharmacy, university of Al-Qadisiyah Assistant prof. D. Bassim Iraheim AL-Shaibani and the head department of Clinical laboratory Lecturer Dr. Ghugran Mohammed Hussein* for providing the facilities required for this study.

I am deeply indebted to every person in my family especially to my father and my mother for their love, helpfulness, moral support, and encouragement they gave me throughout this study.



Dedication

To whom Allah sent as mercy to the worlds....

To the prophet Mohammed

To our diabetic patients in Iraq.....

To my family.....

To everyone I love.....



بَ أَسْلَاحَ أَلْحَ

﴿ يَا أَيُّهَا الَّذِينَ آَمَنُوا إِذَا قِيلَ لَكُمْ تَفَسَّحُوا فِي الْمَجَالِسِ فَافْسَحُوا يَفْسَحِ اللَّهُ اللَّهُ لَكُمْ وَإِذَا قِيلَ انْشُرُوا فَانْشُرُوا يَرْفَعَ اللَّهُ لَكُمْ وَإِذَا قِيلَ انْشُرُوا فَانْشُرُوا يَرْفَعَ اللَّهُ الَّذِينَ أُوتُوا الْعِلْمَ اللَّهُ الَّذِينَ أُوتُوا الْعِلْمَ رَجَاتٍ وَاللهُ بِمَا تَعْمَلُونَ خَبِيرٌ ﴾ ا سورة المجادلة الآية 11



List of Contents

subject	page
List of contents	
Abstract	
Chapter one	
1.1 Diabetes mellitus (D.M.)	7
Chapter two	
2.1 Types of Diabetic	9
2.2 Type I diabetes mellitus or called (Juvenile onset	10
type)	10
2.3 Type II diabetes mellitus or called (Obesity related	10
alabeles)	11
2.4 Gestational diabetes mentus (GDW)	11
2.5 Other types of diabetes	12
2.6 Causes diabetes mellitus	13
2.7 Gene and diabetes	14
2.8 Treatment	14
2.9 Types of insulin	17
2.10 Complications of diabetes is divided into	17
2.10.1 Acute complications include	19
2.10.2 Chronic complications	19
Chapter three	
3.1 Results and discussion	21
Chapter four	
4.1 The conclusions and recommendations	25
5.1 References	26



1.1 Diabetes mellitus (D.M.)

Diabetes mellitus (D.M.) is a common metabolic disorders, is characterized by the disease not to the body's ability to produce or use insulin only, since insulin low in the metabolism of carbohydrates, proteins and fats and the movement of the water affects the ions (Campbell and Recce, 2005) and diabetes are many and complex steep some complications ketoacidosis, which could threaten the lives of patients with diabetes, especially (Type1 insulin-dependent DM,) and some chronic back to winning in the small blood vessels and then affects the three important organs in the body it is the kidneys, causing "renal impairment nephropathy, which ends kidney helplessness, and nerves, causing neuropathy as well as its influence in the eye, causing retinopathy which ends with blindness. have diabetes in large vessels also vessels which leads to many complications, including for the case sclerosis affects atherosclerosis, which is one of the factors leading up to high blood pressure and angina pectoris (Soleimani et al., 2007) and increased rates of diabetes in recent times significantly, with the number of injured is estimated at 176 million people in the world (Soleimani et al., 2007). the number of people with diabetes from 108 million people in 1980 to 422 million in 2014 rose globally diabetes prevalence rate among adults aged over 18 from 4.7% in 1980 to 8.5% in 2014 higher diabetes prevalence rate faster in low and middle income countries. 1.5 million Deaths resulted from diabetes directly while another 2.2 million deaths were attributed to the high level of blood glucose during 2012 estimated happens about half of all deaths due to the high level of glucose in the blood before reaching 70 years of age (WHO,2016). Who expects that diabetes will become the seventh cause of death factor in 2030 (mathers et al.,2006). In Iraq, 2008 statistics confirmed that 10.4 percent of Iraqi



people have diabetes and through experience and our work in this field believe that the ratio is higher and has more than 15% globally as it proved that all three cases diagnosed for it one case undiagnosed this field study was conducted for the purpose of identifying the most important symptoms, causes and the number of children living with diabetes in the province of Diwaniyah and early diagnosis of the disease in order to avoid the serious complications of the disease(WHO,2016).



2.1 Type of Diabetic

The two main known for diabetes type I and type II term has type I diabetes replaces many previous terminology such as gestational diabetes or insulin-dependent diabetes and likewise the second term solution replaces terms such as adult diabetes, obesity-related diabetes, or non-insulin-dependent diabetes apart two types there is an agreed standard naming system for the rest of the species, for example some called the third type of diabetes gestational diabetes pregnant women (ADA , 2004), there is another kind called type 1 diabetes insulin resistance or diabetes is in fact multiple type II diabetes has evolved to become a patient needs insulin injections and adult diabetes is called type is caused by autoimmune latent (2007, Jones) and there is also a diabetes patient infects maturity before reaching the age of thirty, and is a collection of individual genetic disorders accompanied by strong family precedent in cases of type 2 diabetes.

2.2 Type I diabetes mellitus or called (Juvenile onset type)

Risk in this type of approximately 10% of all people living with this disease, this kind of losing absolute insulin autoimmune attack on beta cells or infiltration of lymphocytes T type islets of Langerhans leading to inflammation of these islets, and named insulitis over time leads to immune attack tree smashing beta cells, these appear quickly and unexpectedly when you access the crash to 80 or 90% of beta cells In this case pancreatic fails to control glucose metabolism, requiring the need to give insulin to control and glucose metabolism prevent ketoacidosis coma pH that may arise due to the high level of ketone body (Champ et al., 2005). Of the most important factors to help destroy the beta cells and the emergence of this kind of disease is viral infections, genetic determinants that make beta cells that don't know her body and looks like a ' stranger ' as well as the role of genetics in determining beta cells to



crash this etiology (Guyton and Hall, 2002) there are many tests that can be made to signify autoimmune smashed through the presence of antibodies in infected bodies fluids containing antibodies to cells of the islets itself, and antibodies to insulin, autoimmune antibodies to the enzyme de carboxylase clotamic acid and antibodies to altairosin phosphate enzymes (1 A.2 and 1 A-2 Beta)(Gillespie, 2006).

2.3 Type II diabetes mellitus or called (Obesity related diabetes)

The incidence of this kind are 90% of all people living with diabetes, and differentiate insulin but either not enough or don't respond its receptor in beta cells appropriately to insulin secretion catalyzer.

effect (Jay, 2000) and therefore reduced sensitivity of target tissues as liver, muscle and fat tissue and thinks that one of the main reasons for its occurrence is reduced the number of vectors glucose sensitive transport Insulin (Berger et al., 1989) nor is this type within a group of autoimmune diseases or hereditary and not dependent on insulin, so called non-insulin-dependent diabetes (NIDDM), this disease occurs more frequently among persons who are competent in obese persons over 40 years (Droumaguet et al., 2006) and of the top reasons that lead to the high level of sugar in the genre is resisting the target tissue (liver, adipose tissue, muscle) the normal concentration of insulin, beta cell dysfunction, less resistance when the target tissue susceptibility to respond to the insulin concentration in the blood due to failure to control the production of liver glucose and stay into muscle tissue and fat tissue. (Champ et al., 2005) this kind of diabetes type I hardly know the history of the disease or the disease can remain for many years without showing symptoms such as hunger, fatigue and general weakness and thirst after a while start emerging complications like eye injury and impairment of vascular disorders called kidney, either cardiovascular injuries, vascular disorders



called big and worth mentioning here that the duration of exposure has a big role in the development of such cases (Al-Sa'di, 2005) by another, he found that genetics play an important role in the incidence of this type in addition to other factors which have a significant impact on boot to infection as idle and lack of movement.

2.4 Gestational diabetes mellitus (GDM)

Type II diabetes pregnant corresponds in many respects so similar even in lack of insulin relative weak response of body tissues to insulin (Thomas and Moor, 2005), and between (2-5)% of pregnant women from the disease, but the mother's condition improves or disappears after birth and pregnant gestational diabetes can be cured permanently but it requires medical supervision accurate during pregnancy. but between the (20-50)% of mothers who suffered from gestational diabetes pregnant women can safely second type in later stages of their lives and although the injury is temporary and not permanent but that pregnant diabetes can damage the health of the mother is pregnant, or the health of the fetus and risks to her unborn fetus body inflation increased weight at birth and heart abnormalities, or central nervous system, as well as structural device distortions and jaundice can occur as a result of the destruction of red blood cells (Jones 2007), and in serious cases can prenatal and die that happens in most cases as a result of insufficient nourishment through the placenta due to weak blood vessels and delivery can occur in case of lower function .(Sermer et al .,1995; Growther *et al* ., 2005)

2.5 Other types of diabetes-:

There are many rare causes of diabetes which cannot be classified as a type of first or second or gestational diabetes pregnant women and raises many controversial attempts there classify other types



of diabetes placed many scientists (Kumar et al., 2005) can be summarized as follows-:

1-diabetes is caused by a genetic defect in beta cells, causing loss of functionality. this disease occurs in young people (MODY) maturity onset of M. D. young and is caused by genetic mutations in obtaining nuclear factor 1-Alpha (called MOD. Y3) or 4 Alpha hepatocyte liver cells nuclear factor-4 α signified (MOD. Y1) or nuclear factor 1 alpha-4 beta to liver cells (MOD. Y5) or the enzyme Glucokinase (MOD. Y2) or as a result of genetic mutation in mitochondrial DNA mutation in DNA mitochondria

2-diabetes caused by a genetic defect in the manufacturing process of insulin or do as the imbalance in the conversion of Proinsulin to insulin or mutations in insulin receptor .

3-diabetes is caused by a bug in external pancreatic exocrine secretion defect to the pancreas, such as chronic pancreatitis, iron accumulation in the pancreas leads to destroy tumors and β -cell neoplasia cystic fibrosis cystic fibrosis .

4-diabetes caused by Staphylococcal scarlatina pathies Endocrine disorders like large parties acromegaly, tumors and carcinoma thyroid hormones rise hyperthyroidism and thyroid cancer adrenergic Pheochromacytoma.

5-diabetes caused some injuries such as Rubella virus and cytomegalovirus, Coxsackie virus and Reo type B.

6-diabetes arising from use of drugs and chemical substances such as protease-inhibiter and interferon and thyroid hormone and β adrenergic and glucocorticoides agoinst and thiazide, phenytion 1.



2.6 Causes diabetes mellitus

Basic insulin is a hormone that regulates blood glucose transfer to most cells of the body, especially muscle cells, fat cells but did not move into the cells of the central nervous system and therefore lack of insulin or the body is not responding to any kinds of sugars. turn out most carbohydrates in food into glucose uni in a few hours, this single is the main carbohydrate glucose the blood which is used as energy (fuel) in cells and secreted insulin in the blood by beta cells in the pancreas to lankerhans as a reaction to high blood glucose levels after eating as it pertains to modulate its receptors present in the outer membrane of beta cells and energy gets depolarization and increased calcium entry into the cell, which in turn affects the secretory granules in cytoplasm and then begs insulin outside the cell, and uses insulin from about two-thirds of the body's cells to absorb blood glucose to use as energy for metabolic processes (metabolism) required by the cell to produce other particles or for storage and insulin secretion of insulin from the beta cells (β cell) and reverse conversion glycogen , who works in the opposite direction to insulin and it gets into the blood from the liver glucose while miss muscle cells glycogen conversion mechanism to store glucose. increasing insulin levels to increase in body constructions such as cell growth and increase their number and protein synthesis and storage of fat and insulin is the main in transforming many bidirectional metabolic processes from demolition to construction and vice versa when the blood glucose level is low, it stimulates the burning of body fat. if the amount of insulin available is insufficient, or if the response weak cells insulin (insulin resistance or immunity) would not absorb properly glucose the cells of the body that you need and will not glucose stores in the liver and muscles properly so that the end result is persistent high levels of blood



glucose, poor protein synthesis and some metabolic disorders such as blood pH Acidosis (Kolterman, 1999).

2.7 Gene and diabetes-:

Lead heredity plays a part in patient injury types I and II diabetes and believed that the first type of diabetes is stimulant by some kind of viral infection (Rother, 2007) and other types of stimuli on a small scale such as psychological stress, or stress and exposure to environmental influences such as exposure to certain chemicals or drugs and perform some genetic elements play a role in an individual's response to these stimuli have been tracking these genetic elements found as types of genes related to guidance the white blood cells of any antibodies present in the body it any reliable gene immune cells of the body that you should not attack it from objects you must attack it, though, even for those who have inherited this predisposition to the disease have exposure to environmental stimulus and holds few people living with type I diabetes gene mutated causing diabetes mellitus maturity that affects adolescents and genetics play a bigger role in the incidence of type II diabetes, especially those who have a first degree relatives suffering from the disease are more likely

2.8 Treatment-:

The primary goal of treatment is to maintain the level of blood glucose within the normal level as possible to reduce the incidence of complications associated with the disease and obtaining low or high sugar and unnatural thing in the treatment plan is attempting to regulate blood glucose by eating a healthy diet and maintain a normal body weight (perryll et al., 1996) there are many ways to treat diabetes and modalities the main is :



1-work for perfect weight.

2-sugar diet.

3-out of the organization.

4-medical treatments when needed, which in turn are divided into :

a) chemical treatment.

Chemical treatment:-which in turn is divided into two main areas :

2.8.1 First :- they include insulin which is given by injection under the skin, though his success in curing disease but a number of studies have shown that this treatment short of side effects and does not restrict the development of complications of diabetes (Brownlee and Cerami, 1981) that the insulin used in treatment does not have the same role played by the original hormone (produced in the body) in preventing complications of diabetes, most notably the hardening arteries plus insulin injections with incorrect amounts may not lead to obtain the desired effect, for example using insulin doses lead to very low blood glucose and increased heart rate and mental disorders and may develop case to fainting and death (Bowan and Rand, 1980) and there's the potential for the body's resistance to insulin and when it requires daily doses increase dramatically (Piper, 1986). Either continuous subcutaneous insulin injection pains of using insulin syringeb) treatment plants or natural materials.

2.8.2 second:- they include drugs that usually given by mouth in pill form tablet and divided into

I- **Sulfonylurease Sulfonylurease Meltitinides** – * yoris sulfonyl divides into two parts :

1-the first generation of yoris sulfonyl featuring Acetohexamide and Tolbutamide and Chlorpropamide .



2- the second generation of the yoris sulfonyl featuring Glicazide and Glipizide, Glimepiride and Glibeneclamide and Gliqcazide and. Glibornuide

yoris sulfonyl mechanism relies on the secretion of insulin from the beta cells and reduce the level of glycocane in serum, as well as increasing insulin to link its sensors, the target tissue (Laurence et al., 1973).

2.Biguanides group:

This group includes drug Phenformin and metformine, as observed in 1929 these articles effective hypoglycemic effect sugar antipyretic, then enslaved Phenformine of treatment because it raises the level of lactic acid in the blood as a result of accelerating the process of Glycolysis glucose aerobically leading in the score to the disorders of the gut and general weakness, either metformine it differs from the Sulfonyl ureas not triggered on insulin secretion, reduces the amount of liver made glucose increases the consumption of tissue target glucose, leading to lower blood glucose by inhibition of gluconeogenesis glucose manufacturing process (Marathe et al., 2000) and absorbs a metformine holding primary in the small intestine and metabolized in the liver and not put this medicine through kidney, and common disadvantages are high lactic acid in the blood, liver and gastrointestinal tract, blocking absorption of vitamin B12 (Chan et al., 2001).

3.Alpha-Glycosidase inhibitors

1-Acarbose is sugar Pseudotetrasaccharide microbial origin liar quadruple lead it through or selective competitive inhibition or reversal of α glucosidase enzyme hydrolase, and therefore reduces the absorption of starches and sugars in type II diabetes, as an adjunct to insulin in the first type of illness IDDM, doesn't help insulin secretion from the pancreas and does not work as a surfactant sugar (Gentile et al., 2001)



2-Maglitol is a byproduct of alpha inhibitors glycosidase and derives from 1-desoxynojirimycin and associated inversely with the enzyme Alpha-glycosidase, and completely absorbed from the intestine (Luft, 2001), which is six times stronger than Acarbose in inhibiting absorption of sucrose (Kobavashi, 1999)

2.9 Types of insulin:

1- Fast-acting pur: such as insulin (Aspreu, Haumaloj) fast-in absorption
Starts Work (5-15 minutes) and short-term where continue for (3-4) hours
2- Short –acting pur : such as moderate insulin starts work during half an hour and continue for (5-8 hours)

3-Moderate- acting turbid such as insulin NPH (N) or Lente (L) starts its work during the (1-3 hours) and continues for (16-24)h.

4-Long-acting turbid such as Ultralente (U), (Glargine,Lantus) does not begin its work only after about (4.6 hours) but continue for (48 hours).

5-Insulin mixture(turbid,pure) : two types of insulin mixed in one case such as a mix of moderate and NPH (N) starts its work during the half hour and continue for (16-24) hours and more widely used species.

2.10 Complications of diabetes is divided into-:

2.10.1 A-Acute complications include-:

1- Diabetus Ketonemia

to develop the acetone blood diabetes is severe and serious emergency doubled the lack of insulin to the liver converts fat into acetone bodies used by the brain as fuel (Harper, 1970) but high levels of ketone to instigate against her low (pH) of blood, which causes the appearance of most of the acetone blood acidification symptoms (Myshne , 1967), is characterized by dryness and breathing fast and deep abdominal colic and can be severe and may get lost in advanced stages of the situation and can become severe enough to the acetone blood acidification causes low blood pressure and shock leading to death and urinalysis reveals large



quantities of ketone bodies that come from blood when nominated (Ganong, 1993; McDonald, 1975), and the appropriate treatment to full return to normalcy.

2-Osmosis compression no acetone-:

Coma osmosis coma are double sharp accompanied by many symptoms of blood acidification the acetone but because of the very different treatment when blood glucose level rises above 300 mg/dl pulls water out of the cells into the blood through osmosis, renal excretion and glucose behavior that leads to water and increase blood osmosis if not replace lost fluids by mouth or intravenously osmotic effect to glucose levels with high water to drought, and cells adry body increased as long as the water is taken from them and out of the body through the urethra, and imbalance of body salts and be dangerous for the patient, as it applies to the acetone blood acidification should treatment the situation urgently and especially drought treatment through replacement of lost fluids and depression can evolve into a coma, and is common in type II diabetes than type I. (WHO,2000)

3-Low blood glucose

is a complication of many diabetic medications (Weeker et al., 2003) and can show if patient intake glucose does not cover the treatment of symptoms of the disorder, and sweat profusely elicit autonomic sympathetic nervous system leading to a sense of constant fear and in severe cases lead to coma or even destroy the brain and death for diabetes there are several factors that can lead to low blood glucose, such as excessive use of insulin, or use it at inappropriate times over-in sport or exercise at inappropriate times or lack sufficient intake of food, especially carbohydrates produced glucose and low blood glucose usually results in drug interaction between absolute and relative increase in insulin and weak blood modulate recovery for patients with type I and type II, factors



that play a big role in the prevention or correction of low blood glucose and insulin deprivation is recovery increase glycocan , or absent increase

Morbidity-:

getting diabetes patient sensitivity to infection by bacteria and fungi, and may lead to the emergence of pimples and skin cicatrization urinary Canal injuries that may become increasingly complex and lead to inflammation of the Pelvis kidney, kidney buds necrosis in addition to increase the sensitivity of the patient may increase the chance of infection with tuberculosis in particular type (AL-Egeely, 1999; Weeker et al., 2003)

2.10.2 Chronic complications:

chronic high blood glucose cause damage to blood vessels and cells lining the blood vessels are not dependent on insulin they glucose absorption more than usual, this leads to excessive surface glycoprotein is normal and causes the growth of your name but weaker for vascular cover in case of diabetes is classified this case as a disease of capillaries, the small vessels are damaged and are classified as macrovascular disease because its damage to arteries and this chronic complications can be divided into :

1- Diabetic Retinopathy-:

is diabetic retinopathy (DR) of the most dangerous complications of diabetes incidence rate approximately 40% of patients with type 1 and 20% of patients with type II total for all, up to 25% clinically diabetic retinopathy is classified into three main types (Raman, 2002)

- 1- Back ground diabetic
- 2- Preproliferative diabetic retinopathy (PPDR)
- 3- Proliferaive retinopathy (PDR)



retinopathy leads to blindness and often get the most significant reconstruction and feature large tears and retinal bleeding frequent eyeglass opacity (Laura * Meentyrem, 2004), in the case of proliferative retinopathy characterized this case exciting growth of blood vessels that feature as fragile and unnatural and that grow along the retina and along the surface, be clear gelatin filled the inside of the eye and may lead to blindness.

Diabetic Nephropathy is one of the complications is the most common macroscopic (Janssen, 2002) featuring this case with urinary protein and low efficiency in filtering, arterial hypertension and is highly dangerous cardiovascular injury and death (Kelly et al., 2003 and may result in kidney disease associated with diabetes in death 1993) (Liu et al., that half of the cases of kidney disease in the United States caused by poor kidney resulting from diabetes and diabetic retinopathy diagnosis depends on kidney guides More important, the clinical progression of microalbuminuria appears to albumin in more patients with diabetes especially associated with retinopathy, often shared with hypertension and increased plasma creatinine (A'Amico * Bazzi, 2003)

2- Macrovascular Complications include:

(Weiss and Sumpio, 2006)

1-coronary artery disease leads to angina or myocardial infarction (heart attack)

2-stroke due to lack of circulation.

3- Peripheral vascular disease share in intermittent claudication .

4- diabetic myonecrosis.



3.1 Results And Discussion

Private data taken from diabetes children who starts ages from day to 12 years in the city of Diwaniyah in both birth and children and hospital Hussein and Diabetes Center Specialist Hospital in the general hospital in Diwaniya and for the period from (1 - 9 - 2016) to (15 - 3 - 2017) and took the following information the patient's name, sex, weight, age of the patient, the age of the mother, hyperglycemia, hypoglycemia, the type of medication and dosage, symptoms, complications, reasons in a special form has been organized, as illustrated photo was filling out the form accurately after the meeting doctor competent and the family of the patient and patient the results were as shown table undersigned It has been noted in the table above that cases hyperglycemia more than the incidence of hypoglycemia It was noted that the ages most vulnerable are the reconstruction over five years and was mostly hereditary and there acquired cases and most of the causes shock and there are differences in incidence between males and females did not have the age of the mother influence on the appearance of the disease was noted a decrease in the weights of children with diabetes, the spectrum of the low concentration of insulin and increased glucose concentration in the blood and remains free of the energy cells would receive metabolism of glycogen inventory in the body to compensate for the lost energy and then begins to get metabolize fat save lead to an increased level of fatty acids in the blood stream followed by lipid peroxidation increase as a result of lower energy level and this led to the metabolic processes disorder and the adoption of the cells on the latest alternative energy through oxidation of fatty acids, which leads to the result



to increase the level of ketone bodies and the appearance of the case (Ketosis) with high urea level in the blood, which is considered one of the most important complications seen in children infected as well as the effects of increasing free radicals cause insulin deficiency and increased glucose concentration, which is attributable to him damage made in the liver cells (Dario et al., 1996; Abou Elsoud et al., 2007) and that the high level of sugar resulting from lack of insulin level as a result of damage happening to cells beta Islets of Langerhans this leads to the decline of the role of insulin structural; it works insulin to increase the construction of proteins and lack of decomposition (Selvan et al., 2008) Infection with diabetes accompanied by a decrease in the synthesis of proteins within cells while increasing the level of amino acids in the blood without the benefit of them in protein synthesis (Pain * Garlick, 1974, Yassin et al., 2004). Accompanies the diabetes cases also low concentration of enzymes anti-oxidization substances such as Superoxid dismutase and Catalas in addition to Glutathione responsible for removing the free roots (Selvan et al., 2008) and all the reasons attributed for low weight of when children infected and other symptoms observed when children injured is feeling thirsty and increase the number of times urination investigations as a result of the increase in the concentration of blood sugar, which led to a satisfactory changes in glomerular then arose increase the filtration and hence lose a lot of liquids, leading to increasedperforming dairy cow, that the increase in raising glucose returns to the high level of glucose in blood, resulting from the lack of insulin, where the resulting contains high concentrations of glucose superiority of the susceptibility of the kidney absorbed



in addition to the imbalance in the overall function where at least nomination rate Glomerular filtration In addition to the case of the intransigence of freshly which followed by the overall morbidity diabetic nephropathy, which features blood uremia and therefore increase osmatic pressure on the one hand and the nomination glomerular because of the damage in the glomerular renal syndrome on the other hand, as well as the shortage that may occur in the focus of the ADH may likely in access to the Marks (Bardoux et al., 1999). From another side, the low level of enzymes (dehydrogenases) which play a role in the metabolic processes leading to the fall of the energy level in the body and causing the emergence of clinical signs. In addition to note the emergence of nervous case when children infected and inflammation in the eye and may between (Bishope, 2005) a mechanism for the emergence of eye infections nervous situations the causes of those signs due to the low enzyme degydrogenase and accumulation of Sorbitole in the lens eye retina, liver and kidneys, red blood cell retained placenta were found vesicles sperm and increase the concentration of sugar has caused further accumulation of Sorbitole in neural tissue leading to disable the adriatic-ionic pumps especially cells of showann, which resulted in the result to the access of nervous disorders (Kumar et al., 2003). Other symptoms accompanying the disease was feeling of hunger and the tendency to deal with sugar this is attributable to the diabetes causes reduced power in the body because of low concentration of sugar inside cells especially like hepatic and continue to stimulate the center of hunger in the leg brain (Olivera et al., 2007)



From another side to further focus glycose have negative impacts numerous factors causing glycosylative access some of the proteins, including amino acids and glycogen causing precipitation in cells lined blood capillaries in glomeruli and padded reviewing Bowman and thus increase thickness, making it were not competent in keeping some of the articles in the blood plasma and then leaked to the urinary tubular, in addition to this the glucose increase in blood, and entry to the cells all this lead to satisfactory changes as a result of to increase the permeability of the blood capillaries causing deposition of plasma proteins in the gap between the blood capillaries (Evans et al., 1995; Bartosikova et al., 2003) and the decline in the level of Glucokinase enzyme responsible for the fumbled beta cells high levels of glucouse with low Hexokinase Phosphoglucoisomerase, increase the level of G-6-Phosphatase) (Ragavan & Krishna Kumar, 2006) that diabetes may be accompanied by a significant increase in the toxic effects resulting from the increase in the free roots in the body. It has been found that increase blood glucose leading to the lack of balance in the Redox inside cells especially in the liver (Galluo et al., 1993), and thus to increase those roots that lead to imbalances in the functions of those cells and access damage oxidative membrane with increased opportunity fatty oxidation (Vallabhji et al., 2001) and the most important treatments used when children injured, insulin two type (pure and turbid) where there are types of the insulin.



4.1 The conclusions and recommendations

We conclude of this field study conducted in hospitals in Diwaniyah children diabetes cases when children grow significantly in the city and the bad use of correct and lead to deaths among the injured and some families to the use of the wrong herbs led to serious complications at the child and that most cases were hereditary and was treating the disease depends on insulin and recommend the following.

The failure of marriage as much as possible between relatives in families with diabetes and got marriage must be child control because the early detection of the disease is very necessary to limit the access of complications.

1-Monitoring of the Child, particularly when families who were carrying the disease since birth through the periodic examination of the focus of blood glucose

2-Monitoring of the child if any of the symptoms of the above-mentioned in the search

3-Reduce the sugars when children who have getting ready diabetes

4-An attempt to families as far as possible not to endanger the child for any kind of shock

5-Reduction of provoking terror and concerns of children that may cause a shock when boys

6- the commitment of dates of the periodic review of the doctor paths to children infected with the disease and treatment.



5.1 References

1-Abdel-Wahab, et_al. (2002). Vitamin C supplementation decreases insulin resist Health benefits and practical aspect of high fiber diets ance and improves glucose homeostasis in obese hyperglycemic mice. Metabolism; 51: 514-7.

2-Anderson, et_al .(1995).. AM. J. Clin. Nutr. , 59:42-47.

- 3-A.H.M.et_al.(2000). De creasing dietary fat saturation lowers HDI-C and increases hepatic HDL binding in hamsters. British J. Nutr., 83:151-159.
- 4-AL-Karagoly,and H. K. (2007). Clinlopathology study of Experimental induced diabetes mellitus domestic Rabbits. M. SC. Thesis of Collage of vet. Medicinal /University of Busrah.
- 5- Abbey , M.;et_al. (1994).partial replacement of saturated fatty acids with almond or walnuts lower total plasma cholesterol and low density – lipprotein cholesterol . Am. J. Clin . Nutr., 59:995-999.
- 6-Brufau, G.et_al. (2006). Nuts : source of energy and macronutrients . Br. J. Nutr., s45-s51.
- 7- Borch-Johnsen, et_al. (1984). "Relation between breast-feeding and incidence rates of insulin-dependent diabetes mellitus. A hypothesis ."Lancet .6–1083 :(8411) 2 doi/10.1016:S0140-6736(84)91517-4 .PMID6150150 .
- 8- Coggeshall, J,et-al. (1985). Biotion Supplementation improves glucose and insulin tolerances in genetically diabetic kk mice life Sci; 42: 1323-1330.
- 9- Criqui, M.H. and Colomb , B.A. (1998). Epidemiologic aspect of lipid abnormalities . Am. J.Med., 105(IA):85-75.
- 10- Chan, J.K.et_al . (2001). Dietary linolenic acid is as effective as oleic acid and linoleic acid in lowering blood cholesterol in normolipidemic men . Am. J.Clin . Nutr., 53:30-34.



- 11- Elin,R.J. and Hosseini , J.M.(1993). Is the magnesium content of nuts a facter for coronary heart disease . P.Arch . Intern. Med . 153:779-780.
- 12- Farquhar,and J.W. (1996). Plant sterols : their biological effect in humans . In spiller , G.(ed): handbook of lipid in human nutrition . Boca Roton: CRC pres, Pp:107-112.
- 13- Goel, V.; et_al.(1997). Cholesterol lowering effects of rhubab fiber in hyper cholesterolemic men . J.AM. College of Nutr., 16:600-604.
- 14-Gomori, G. and Goldner , M.G. (1943). Production of diabetes mellitus in rats with alloxan . proc .Soc. Exp. Biol. Med., 54:287-290.
- 15- Galletto, R.; et_al. (2004). Absence of Antidiabetic and hypolipidemic effect of Gymnema sylvestre in non diabetus and Alloxan diabetic rats-Brazilian Archives of Biology &Technology; 47: 545-551.
- 16-Global report on diabetes. World Health Organization, Geneva, (2016).
- 17- Hyppönen ,E.; et_al. (2001). "Intake of vitamin D and risk of type 1 diabetes: a birth-cohort study ."Lancet .1500 :358 doi/10.1016:S0140-6736(01)06580-1 .PMID11705562 .
- 18- Jamal, G. A. (1991). Pathogenesis of diabetic neuropathy: the role of the n-6 essential fatty acid & their Eicosonoil derivatives. Diabet. Med., 7: 384-369.
- 19- Jambazian, P. R.;et_al.(2005).Almond in the diet simultaneously improve plasma alph- tocopherol concentration and reduce plasma lipids J.Am. Diet. Assoc .105(3):449-454.
- 20- Jenkins, D.J.; et_al. (2002). Dose response of almond on coronary heart disease risk facters: blood lipids, oxidized low density lipoprotian (a), homocysteine, and pulmonary nitric oxide :a



randomized, controlled , Crossover trial .Circulation, 106(11)1327-1332.

- 21-Jenkins, D.J.; et_al.(2008). Almond reduce biomarkers of lipid peroxidation in older hyperlipidemic subjects.J. Nutr.138:908-913.
- 22- Kjeldsen, S.E.; et_al. (2006). "Effects of valsartan compared to amlodipine on preventing type 2 diabetes in high-risk hypertensive
- 23-Knowler, W.; et_al. (2002). "Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin ".N Engl J Med 403–393 :(6) 346.
- 24- Kumar, V.; et_al. (2003). Basic Pathology. 7th Ed. Saunders . Philadelphia, Pp: 642-647.
- 25- Lenich, A. C.; et_al. (1991). Effect of dietary cholesterol and alloxan diabetes on tissue cholesterol and apolipoprotein EMRNA level in th rabbit. J. Lipid Res., 32(3): 432-438.
- 26-Lindström, J.; et_al. (2006). "Sustained reduction in the incidence of type 2 diabetes by lifestyle intervention: follow-up of the Finnish Diabetes Prevention Study .".Lancet .9–1673 :(9548) 368 doi/10.1016:S0140-6736(06)69701-8 .PMID17098085 .
- 27- Mayes , P.A.(2002). Structure and fuction of the lipid- soluble vitamine Structure and fuction of the water- soluble vitamineand pantose phosphate pathway and other pathway of hexose metabolism . in : Harpers Biochemister , 25th ed, Appleton and Lange, Stanford , Connecticut , Pp:219-223, 640-649.
- 28- Mendez, et_al. (2005). L- arginine and polyamine administration protect B- cell against alloxan diabetogenic effect in Sprague-Dawley rats. Biomed pharmacother ., 59:283-289.



- 29-Mir, S. H.; et_al . (2008). Biochemical & histomorphologjcal study of streptozotocine induced diabetes mellitus in rabbits. Pakistanian Journal of Nutrition., 7(2): 359-364.
- 30-Murray, R.; et_al. (2000). "Harper's biochemistry" 5 th edd., Appleton and Lange. U.S.A. pp doi/10.1056:NEJMoa012512 .PMID . 1183252730- Naim Shehadeh, Raanan Shamir, Moshe Berant, Amos Etzioni (2001")Insulin in human milk and the prevention of type 1 diabetes ."Pediatric Diabetes .177–175 :(4) 2 doi/10.1034:j.1399-5448.2001.20406.x
- 31- Naim Shehadeh,et_al. (2001")Insulin in human milk and the prevention of type 1 diabetes ."Pediatric Diabetes .177–175 :(4) 2 doi/10.1034:j.1399-5448.2001.20406.x
- 32- Oakenful, D. and Sidhu, G.S. (1999). Could saponins be a useful treatment for hypercholerterolemia? Eur. J.Cli. Nutr., 44:79-88.
- 33-Plants database .(2006). United States department of agriculture . Natural Resourcs Coservation service . Prunus dulcis (P.Mill).
- 34- Plessi, M.;et_al. (1999). Dietary fiber and some elements in nuts and wheat brands, Food Comp . An., 12:91-96.
- 35-Prince, D. S.; et_al. (2004). Antidiabetic & Antihyperlipidemic of alchoholic syzigum cuminiseeds in alloxan induced diabetes albino rats. J. Ethanophamaol., 91(203): 209-213.
- 36- Projections of global mortality and burden of disease from 2002 to 2030. Mathers CD, Loncar D. PLoS Med, 2006, 3(11):e442.
- 37- Ramesh, B.; et_al. (2007). Protective effect of umbiliferone on membranous fatty acid composition in streptozation-induced diabetic rats. European. Journal of Pharmacology., 566(1-3): 231-239.
- 38-Ravivijayavargia, V.; et_al.(2000). Hypoglycemic effect of aqueous extract of Enicostemma littoral Blume (Chhotachirayata) on



alloxan induce diabetes mellitus in rats , Indian .J. Exp. Biol., 38:781-784.

- 39- Skyrme-Johnes, et_al. (2000). Vitamin E Supplemention improves endothelial function in Type I diabetes mellitus: arandomized, Placebo- controlles, Study. JAM coll cardiol;36: 94-102.
- 40-Sliva-Sousa, Y. T.; et_al. (2003). Enamel hypoplasia in a litter of rats which Alloxan induced diabetes mellitus. Braz. Dent. J. 14(2): 65-69.
- 41-Spiller, G.A. (1997). Effect of plant –based diets high in raw or roasted almond or roasted almond butter on serum lipoprotein in humans. J.Am. coll.Nutr., 22(3):195-200.
- 42-Steel, R. G. T. and Torrie, J. H. (1980). Principles and procedure of statistics. McGraw-hills, USA
- 43- Stuebe, A.M.; rt_al. (2005). "Duration of lactation and incidence of type 2 diabetes ."JAMA .10–2601 :(20) 294 doi/10.1001: jama. 294.20.2601 . PMID16304074 .
- 44-Teixeira, F. D.; etal. (2006). The efficacy of folk medicine in the management of type 2 diabetes, mellitus: results of randomized controlled trail of syzygium cumini (L). skeels.
- 45- Tierney,LM.; et_al. (2002).Current medical diagnosis and treatment. International edition.New York:Lange Medical Books/McGraw-Hill (1203-1215.
- 46- Valsta, L.M.; et_al. (1995). The effects , on serum lipoprotein levels of two monounsaturated fat rich diets differing in their linoleic and linolenic acid contents . Nutr. Metable. Cariovase . Dis., 5:129-140.



- 47-Virtanen, S.;and Knip, M. (2003). "Nutritional risk predictors of beta cell autoimmunity and type 1 diabetes at a young age ."Am J Clin Nutr .67–1053 :(6) 78 PMID14668264.
- 48- Wasko ,M.C.; et_al (2007) Hydroxychloroquine and risk of diabetes in patients with rheumatoid arthritis ."JAMA .93–187 :(2) 298 doi/10.1001:jama.298.2.187 .PMID17622600 .

