

1.1 Introduction

Diabetes mellitus is a common medical problem having a significant morbidity and mortality. It has a genetic predisposition, although environmental factors play their role in its genetic expression. Like many other inherited traits, blood groups are also genetically predetermined and therefore may have an association with diabetes mellitus. Identification of a positive association with blood groups might reflect increased susceptibility to and a negative association protection against diabetes mellitus (Waseem *et al.*,2011).

Ever since the discovery of blood groups in 1900, there have been efforts to discover a possible association between ABO and Rh blood groups and different diseases. The data obtained from studies in patients with gastric cancer, salivary gland tumors, duodenal ulcer, colorectal cancer, thyroid disorders, ovarian tumors, small cell carcinoma of lung and coronary heart diseases have shown an association with ABO blood groups. This information has led to the assumption that some other diseases might also be associated with ABO and Rh blood groups. Such associations may have significance to identify susceptibility to diseases and adopt possible preventive measures to decrease the prevalence (Waseem *et al.*,2011).

1.2 Literature review

1.2.1 Physiology of blood:- Blood is specialized connective tissue which circulates in a close system of blood vessels, The average individual has approximately 70 ml of blood per kilogram body weight (70ml/kg).approximately 50-60% ,The liquid component is called plasma and nearly 90% is water,The remaining are protein and other compounds,Serum is a part of whole blood without addition of anticoagulant ,it is essential as plasma except clotting factor and fibrinogen has been consumed(William ,2002).

1.2.1.1 Blood cell and function of blood:-

1- Erythrocyte is (red blood cell)

2- Leukocyte is (white blood cell)

3- Thrombocyte is (platelets)

-The function of blood is the exchange of respiratory gases ,transport oxygen from the lung to tissue and delivers carbon dioxide from tissue loading the lungs to be exchanged and excreted, Blood also transport metabolic wastes to the lung ,kidney ,skin and intestine from removal,

Blood is also responsible for maintaining acid base balance(William2002).

1.2.2 **Blood group system :**

Approximately 400 red blood cell group antigen have been described the clinical significance of blood group in blood transfusion is that individual who lack a particular blood group antigen may produce antibody react with that antigen. (Daniels and Bromilo, 2007).

1.2.2.1 Discovery of blood groups:-

Before 1901 the prevailing belief was that all human blood was same. However this changed in 1901 with Karllandstiener landmark the discovery of ABO blood groups.

Landsteiner an Austrian immunologist , noticed that human blood mixed in a test tube with other specimens of human blood sometimes resulting in agglutination by incubating red cell from same individual with serum from other ,the scientist identified agglutination pattern leading to the intial identification of blood group A , B and C(C was later named O). in 1902 Alferd Decastello and Adrino Sturli two of Landsteiner`s former students ,discover AB an afourth blood group (Hillyer *et al*, 2007).

1.2.2.2 Antigens:-

Antigens are substance which could be proteins or non proteins and when introduced to animal host can cause

production of specific antibody thereby inducing an immune response, cellular or humoral or both, The antigen can react with elicited antibody or with sensitized lymphocyte to induced immune response, physical property are governed by criteria's such as site of antigen ,number of antigenic site ,antigenic determinant, site of antigen is determined by cell structure , size , shape , and membrane condition in blood group ABO antigen are extra membranous where Rh antigen are assumed to be intra membranous , number of antigenic site and closeness of these site explain the diversity in behavior of antigen by mean of immune electron microscopy it has been confirmed that ABO antigen occurs in cluster whereas Rh antigens are isolated and estimated that 800,000.Antigenic determinant is the particular site of an antigen molecule that combine with specific antibody Specificity is also a very important property of antigen for which polysaccharide component of antigen is responsible for basis of dividing human blood group in to A;B;O;and AB ,Antigenicity gives as an idea about the potency of particular antigen, reticulocyte are strongly agglutinated by anti A anti B (Talib and Dutta,1995)

1.2.2.3 Antibody ;-

Agglutinin or antibody are serum protein more specifically immunoglobulin ,In human being antibodies can be natural or immune depending on its origin or source,Natural antibody can be due bacterial or plant origin because it has been

observed that both may contain A and B antigen and as result of immunization produce anti A or anti B Of IgM type, antibody in new born infant -normally human fetus dose not began to produce significant amount of antibody when in mother womb,Usually IgM and after birth since the IgM dose not cross placenta in the cord serum IgG is usually not detected and if at all then this can be attributed to placental transfer antibody starts appearing in second month of intrauterine life the IgM Reachs adult level by 6 months to 1year (Talib and Dutta,1995)

1.2.2.4 Nature of blood groups:-

The term blood group is usually restricted to blood cell surface antigen ,and generally to red cell surface antigen and generally to red cell surface antigen it was genetic variation in human red cell membrane protein, glycoprotein and glucolipids These variation are detected by alloantibodies,which occur either naturally as a result of immunization by ubiquitous antigen present in the environment or as a result of alloimmunization by human red cell (Daniel, 2002).

1.2.2.5 Blood group inheritance:-

Almost all blood group genes are expressed as co dominant antigens i.e. both genes are expressed in the heterozygote, some blood group genes has been assigned to specific

chromosome, eg ABO system on chromosome 9 Rh on chromosome 1 (Lweis *et al.* ,1996).

1.2.2.6 Clinical importance of blood group:-

Blood groups are of great clinical importance in the blood transfusion and transplantation. In fact the discovery of ABO system was one of the most important factor in making practice of blood transfusion possible, Many blood group antibody are have the potential to cause rapid destruction of transfused red cell bearing the corresponding antigen giving rise to hemolytic transfusion reaction (HTR) either immediately several days after transfusion (Daniels and Bromilo ,2007).

1.2.2.7 Clinical significance of blood group antibody:-

In immunohematology ,antibodies may be classified as naturally occurring or immune antibodies .this means that antibody molecules may be present in individuals regardless of that fact that there has been no known stimulus such as transfusion of antigen different blood or fetomaternal hemorrhage.both naturally occurring and immune antibodies can be of importance in immunohematology antibodies can be further divided into categories (alloantibody` which has specifically against an antigen present in the individual own red cell (Daniels and Bromilo ,2007).

1.2.3 The ABO blood group System :-

1.2.3.1 Discovery ABO System:-

ABO blood group system is the most important one with respect to blood transfusion hemopoietic stem cell transplantation and solid organ transplantation , Karl Landsteiner was the first who discover human alloantigen by using conceptually simple experiment (Simon *et al* ,2009)

1.2.3.2 Structure of ABH and related antigens

The ABO blood group system consist of 2 antigens A and B the indirect

product of the A and B ,threr are four phenotype A,B ,AB , and O.

A phenotype results from genotype A/A or A/O , B phenotype results from B/B or B/O ,the AB phenotype from A/B ,O phenotype results from genotype O/O ,although many variations of ABO phenotypes exist , almost all basically quantitative modifications of the A and B antigen (Daniels and Bromilo ,2007).

Table 1.2.

ABO antigen ,antibody and genotype adapted from (Daniels and Bromilo ,2007).

ABO group	Ag in RBCs	Ab in serum	genotype
O	Non	Anti A,B	O/O
A	A	Anti B	A/A,A/O
B	B	Anti A	B/B,B/O
AB	A and B	Non	AB

1.2.3.3 ABO gene:-

ABO gene is located on the long arm of chromosome 9 , comprises seven exons and encode protein with structural characteristic of glycosyltransferase product of the A and B alleles differ by four amino acid encode by exon 7 two of which determined whether the enzyme product has GalNAc transferase (A) or Gal- transferase (B) activity ,the majority of O alleles (called O') resemble to A₁ ,but have a single base deletion in exon 6 ,which create a shift in the reading frame and scramble the amino acid sequence after the first quarter of the transferase polypeptide ; introduction of premature stop codon truncates any putative polypeptide about 3% of the O allele (called O'') have a single nucleotide polymorphism (SNP) that changes one of the vital amino acid in the catalytic site, inactivating the enzyme, the A₂ allele has a single base deletion immediately before the usual termination codon , creating a reading frame shift and abolition of this stop codon, This creates an A- transferase with extraneous 21 amino acid on the C terminus , which accounts for its reduce efficiency as GalNc transferase A₁,A₂,B,O₂ genotypes can be identified by polymerase chain

reaction (PCR) with allele specific primers or followed by analysis with restriction enzyme (Hoffbrand *et al* , 2005).

1.2.3.4 H genes

FUT1,are responsible for production of H antigen, both encode α 1-2 fucosyltransferase that catalyzed the transfer of fucose to terminal galactose residue of the H precursor chain FUT1 is active mesodermally derived tissue including hemopoietic tissue ,and responsible for H expression on red cell, homozygosity for inactivating mutation in FUT1 gives rise to Bombay and related phenotypes. FUT2 is responsible for H expression on endodermally derived tissue ,including those responsible for secretion and hence is the gene responsible for ABH secretion, secretor are homozygous or heterozygous for an active allele (Se)at FUT2, non -secretor are homozygous for an active allele (se) (Hoffbrand *et al*, 2005).

1.2.3.5 Secretion status of blood grouping:-

Almost everybody expresses H antigen on their red cell, but only about 80% of European have H antigen in their body secretion; these people are called ABH secretors because, if they have an A and/or B gene ,they also secrete A and /or B antigen ,the remaining 20% are called ABH non secretor as they do not secrete H,A,or B (Daniels,2002)

1.2.3.6 Sub group of ABO Blood group system:-

1.2.3.6.a Sub group A₁ and A₂

A₁ and A₂ in European about 80% of group A individuals belong to subgroup A₁ almost all the rest being A₂ ,The distinction is most conveniently made by testing red cell with the lectin form *Dolichos biflorus* when diluted appropriately the lectin agglutination only A₁ cell (Klein and Anstee , 2005).

1.2.3.6.b Sub group of B

Sub group B appear to be much rare than weak a subgroup, although this probably reflects the relatively low frequency of B gene in many population (Daniels ,2002).

1.2.3.7 Bombay phenotype :-

In 1952 Bhend described the abnormal blood groups of three men from Bombay whose red cells were group O but H negative , all had anti H in their serum .this rare phenotype later become known as Bombay or Oh phenotype (Daniels , 2002).

Individuals with rare Bombay phenotype are homozygous for inactive H gene (h\h), their red cell are not agglutinated by anti A or B regardless of ABO genotype but are not group O as they are also not agglutinated by anti H. The serum of Bombay subject contains anti H, anti A and\or anti B, parents

and offspring of Bombay individuals, who are heterozygous for the inactive H allele (H/h) have H and red cell of normal ABO phenotype (Hoffbrand *et al*,2005).

1.2.3.8 ABO antibody

1.2.3.8. a Anti H

Anti H antibody detects the precursor of A and B antigens, it's characteristically agglutinate group O and A₂ more strongly than A₁ and B cell (Daniels, 2002)

1.2.3.8. b Anti A

Group A specificity has been found since in a number of seed including *Dolichos biflorus*, an extremely useful blood grouping reagent because it agglutinate A₁ cell far more readily than A₂ cells and so ,when appropriately diluted, distinguishes A₁ and A₁B from A₂and A₂B (Daniels,2002).

1.2.3.8.c Anti B

B specific lectins are less abundant than A specific lectins , they are found together with anti H ,in the arils (seed Goat) of various specieses of *Evonymus* in the fungus fomes fomentations and in the sea weed *ptilota plumoxa* (Daniels , 2002)

1.2.3.8.d Anti A,B

Several seed extracts agglutinated A and B cells but not O cell, in some cases this may be because of one lectin cross reacting with both A and B structure, BSI, one of at least three lectins in *Bandelraea simplicifolia* seed, comprise five isolation made up of different proportions of two subunits (Daniels, 2002).

1.2.4 Rheuses blood group :-

The Rh system is a second to the ABO system in important in transfusion practice because Rh antigen, especially D, are highly immunogenic and cause hemolytic diseases of the fetus and newborn (HDFN) (Simon *et al* 2009), Rh is the most complex blood group system comprising 46 antigen numbered RH1 to RH53 with seven number's absolute. The Rh antigen are encode by two homologous, closely linked gene in the short arm of chromosome 1 (Daniels, 2002) 49 specificities. The most important of theses is D, and then C, c, E, e (Murphy and Pamphilon 2005).

Rheuses antigens are only expressed on red cell, but they are not present in body fluid (Cheesbrough, 2006)

According to Daniels In 1939 Levine and Sietson, investigated a hemolytic reaction which resulted from the transfusion of a women with blood form her husband Levine Sietson showed that this new antigen, which they did not name, was independent of the known blood group, ABO, MN and P. They suggested that the mother had been immunized

by a fetal antigen of paternal origin ,and that hemolytic episode was caused by maternal antibody reacting with that antigen of paternal origin and that the hemolytic cell, the Rh blood group system was discovered in 1940 Landsteiner and Wiener, they made an antibody by injecting Rhesus monkey red cell into rabbits , the antibody was called anti Rh agglutinated rhesus monkey red cell (Daniels ,2002)

Lavine and Sietson explained the cause of many unexpected transfusion reaction .in 1945 Coombs, Mourant and Race described the use of anti-human globulin sera to detect IgG antibodies in compatibility testing ,thus previously the still used coombs test (Hillyer *et al* 2009).

1.2.4.1 Nomenclature and genetic model

The Fisher-Race nomenclature suggested that three closely linked genes (C\c. E\e and D) while the winer nomenclature (Rh-H1) was based on his belief that a single gene encode one “agglutinate” that carried several blood group factors (Simon *et al* 2009).

1.2.4.2 Rh antibody

Rh antibodies are usually produce in response to red cell immunization resulting from blood transfusion or pregnancy, although ‘ naturally occurring’ Rh antibodies are not unkown Rh antibodies react optimally at 37 °C (Daniles 2002).

1.2.5.5anti D

D is the most immunogenic antigen and the most clinically relevant, thus it is the Rh D antigen of the Rh blood group system is one not able exception. As many as 80% of Rh D negative patients exposed to Rh D positive RBCs may develop high titer ,high affinity ,anti Rh D IgG antibodies that may persist for the rest of their live ,even if they are never exposed to the antigen again .The resulting antibodies can cause hemolytic transfusion reaction and can cross the placenta , causing hemolytic diseases of newborn, when present in Rh D negative woman carrying an Rh D positive fetus (Hillyer *et al*,2001).

1.3 Diabetes Mellitus

_Blood glucose concentration are maintained within very close limit very close limit in healthy people, Normal blood glucose concentration 4.5 _5.5 m mol\l , 5% of glucose concentration in healthy people increase after meal.

Reduction in glycaemia are produced by sever sudden un accustomed exercise or prolong fasting ,Is the most common metabolic disorder (William *et al.*,1995).

Diabetes is chronic disease, which occurs when the pancreas dose not produce enough insulin or when the body can not

effectively use insulin it produced .this lead to an increased concentration of glucose in the body(WHO , 2006).

Diabetes mellitus are group of metabolic disease in which a person has high blood glucose because the body does not produce enough insulin or because cell do not respond to insulin that is produced (lawernce. *et al*,2008) .

1.3.1Type of diabetes :-

1.3.1.1 Type 1 diabetes mellitus:-

Referred to insulin dependent DM or juvenile diabetes (lawerance 2008)

Approximately 5-10% of all diabetic patients and these patients require more medicattention than non insulin dependent patient, IDDM is characterized by severe insulin deficiency state consequent upon beta cell destruction (William *et al*,1995).

The degree of insulin deficiency is so severe that this patient requires exogenous insulin therapy to avoid ketoacidosis.

Typically IDMM results from autoimmune destruction upon the pancreatic beta cell. The attack is a marked systemic indices of autoimmune process such as circulating islet cell auto antibodies and change in circulating B lymphocyte subsets. The process of beta cell destruction usually takes many months and occurs in cycle deterioration and remission (William *et al*,1995).

1.3.1.1.a Genetic susceptibility:-

Certain genetic markers are associated with high prevalence of IDDM, Most of these genetic a marker are found on chromosome 6` in gene related to histocompatibility linked antigen (HLA), Most IDDM is associated with HLA-DR₄ present in childhood whilst that association with HLA-DR₃ has more variable age of onset (William *et al*, 1995).

1.3.1.2 NON - Insulin-dependent diabetes mellitus (NIDDM):-

NIDDM is most commonest form of diabetes mellitus in western societies.

Its often considered a diagnosis of exclusion, NIDDM is probably not single condition.

In all NIDDM patients there is insulin resistance and relative insulin deficiency (William *et al*,1995).

Molecular biological techniques have not yet found NIDDM to be consistently associated with any abnormalities of the DNA coding of insulin Abnormality in glucose gene have been shown to cause some cases of MODY type diabetes but not typical NIDDM (William *et al*,1995).

1.3.1.3 Gestational diabetes:-

Gestational diabetes is first recognized during pregnancy(WHO ,2006).

Gestational diabetes mellitus (GDM) resemble type 2 Diabetes in several aspects involving a combination of relative inadequate insulin secretion and responsiveness, it occurs in about 2_5% in all pregnancies and may cure or disappear after delivery (Lawrence, *et al.*, 2008).

Untreated gestational diabetes can affect the health of the fetus and mother . Risk to baby include macrosomia (high birth baby), congenital cardiac and nervous system abnormalities other type are pre indication condition that occur when a person blood glucose level are higher than normal but donot higher enough for diagnosis of type 2 diabetes (Kehuda , 2009).

1.3.1.4 Mason/MODY type diabetes :-

Family studies have revealed a range of variant of diabetes most of this variant of NIDDM, One of most NIDDM subtype known as Mason (after apropositus) Or MODY(maturity onset diabetes of youth). This variant is characterized by onset of NIDDM in late teenage or early twenties, Clinical feature include slow progression to insulin requirement and a reputatation for relative lack of tissue damage in some families (William *et al*, 1995).

1.3.1.5 Secondary diabetes:-

There are several other diseases which may cause diabetes or glucose intolerance, Most of these are readily diagnosed

perhaps with haemochromatosis and chronic pancreatitis as the least readily apparent (William *et al* ,1995).

1.3.2 Signs and symptoms of diabetes mellitus:-

The classical symptoms of diabetes mellitus are polyuria, polydipsia, and polyphagia, prolonged high blood glucose causes glucose absorption which lead to change in the shape of lenses of eye resulting in visual change :Blurred vision is common complain leading to diabetes diagnosis type1

cases of rapid vision change but type 2 is more gradual (Cooke and Dlotnic,2008).

1.3.3 Diagnosis of Diabetes Mellitus :-

-DM is characterized by recurrent and persistent hyperglycemia and is demonstrated by the following :

-fasting blood glucose level ≥ 126 mg/dl.

-2 hours after 75gram oral glucose load in tolerance test ≥ 200 mg/dl.

Positive results are confirmed by repetition of the above method several days in the absence of glycemia (Kehuda , 2009).

Individuals with fasting glucose level between 100-125mg/dl are considered to have impaired fasting glucose. Patients with plasma above 140mg/dl but not over 200mg/dl 2hours after 75gram oral glucose load are considered to have impaired

glucose tolerance, of these 2 pre-diabetic state the later in particular is major risk factor for progression to full blown DM (Nathan *et al.* ,2005).

1.3.4 Complications of diabetes mellitus:-

1.3.4.1 Acute Complication of DM:-

1-Severely elevated blood glucose level due to lack of insulin or relative deficiency of it which lead to:-

In type 1: occur either glucose urea which results in loss of fluid and electrolyte in urine or cause in ability to store fat and protein along with breakdown of existing fats and protein stored which result in ketoacidosis and release ketose in blood which turn into acidic which called DKA (diabetesketoacidosis), have many symptoms such as nausea, vomiting, and abdominal pain which can be developed a shock, coma, and event death (Nathan *et al.*,2005).

-In type2 diabetes: stress and medication eg;corticosteroid if elevated may lead to hypersmolar state(increase blood osmality).which lead to hyperosmlar coma which occur in elderly patients.

2-abnormally low blood glucose level due to too much insulin or other glucose lowering medication can lead to central nervous system symptoms such as confusion ,dizziness,weekness and if glucose level less than 65mg/dl occurred irreversible brain damage. (Nathan *et al.*, 2005).

1.3.4.2 Chronic complications of diabetes mellitus :-

1-diabetic retinopathy (eye complication).

2-diabetic nephropathy (kidney complication).

3-diabetic neuropathy (nerve complication)(Nathan *et al* , 2005).

1.3.5 Management of diabetes:-

DM is a chronic disease difficult to cure. Management of diabetes concentrate need to keep blood glucose level as close to normal (Euglycemia) as possible without presenting patient danger. This can be achieving by dietary management exercise and appropriate medication ,insulin only for type1DM, and oral medication for type 2patient education understanding and participation is vital which make the complication of diabetes are less common and less sever in who have well managed blood glucose level (Pignene *et al.* ,2010)

1.3.6 Diabetes mellitus in Sudan:-

The prevalence of DM in Sudan, as in many low income countries, is increasing to epidemic proportion, leading to the emergence of public health problem of major socio-economic impact.

Before 1989 all knowledge about DM in the Sudanese population was based on few hospital based study ,but later

a series of investigation explored epidemiology and characteristic of the disease in collaboration with Uppsala university ,Sweden (Abdelgader,2004).

1.4 previous studies :-

Previous studies showed different results. A study in India by Sharma (2013) showed no association between ABO blood group and both types of DM (1 and 2) and B is most frequent blood group, , AB in the study of Sharma *et al.*,(2014) was least common blood group.

Jassim (2012) in Iraq reported that the O blood group is the most frequent group among diabetic patients, and Jassim reported that blood group AB is least common.

Negative association between A and O blood group was reported by Kamil *et al.*,(2010) in Malaysia and no association between DM and blood group B and AB least blood group is AB.

1.5 Rationale:-

A number of studies were conducted to investigate association between ABO blood group system and some diseases condition. Some of these studies reported significant Association , suggesting that ABO Blood group have an impact on the infection status of the individual Possessing a particular ABO Blood group other study determined .

Study done in United States showed that The frequency of blood group A was statistically significantly higher amongst pancreatic cancer patients compared to its frequency amongst the regional blood donors (Julia *et al.*,2010)

1.6 Objective

1.5.1 General objective:-

Association between ABO blood group and Rh factor and diabetes mellitus.

1.5.2 Specific objective :-

_To correlate frequency of ABO blood group and type 1 and type2 diabetes mellitus according to age and gender.

_ To correlate frequency of Rh factor and type 1 and type2 diabetes mellitus according to age and gender.

Chapter two

Materials and methods

2.1 Study design:

Cross sectional study to determined association between ABO blood group and Diabetes mellitus in period from 17 march 2014 20 may2014.

2.2 Study area:

The study was carried out in Military Hospital and Ahmed Gassim paediatric hospital in Khartoum

2.3 Study population:

Patient affected by diabetes mellitus from different age and gender in Military hospital and Ahmed Gassim hospital.

2.4 Inclusion criteria:

All patient who diagnosed as diabetic patient in Military hospital and Ahmed Gassim hospital in period between 17 March 2014 20 May 2014.

2.5 Exclusion criteria:

People not diagnosed as diabetic patient in Military hospital and Ahmed Gassim hospital in period between 17 March 2014 20 May 2014

2.6 Ethical consideration:-

Approval of ethical committee of the faculty was taken, Informed consent was obtained from all participant in the study.

2.7 Collection of blood sample :-

One ml of blood were either (capillary or venous) collected in tri sodium citrate anticoagulant .

Capillary blood for type1

Venous blood for type 2

2.7.1 Collection of capillary blood in type 1 DM

_with cotton wool dipped in 70% alcohol the tip the third finger was cleared pricked firmly and rapidly the first drop of blood was wiped and collect one ml.

2.7.2 **Collection of venous blood in type 2 DM**

_with cotton wool dipped in 70% alcohol the for arm select large clear vein enter the vein with single puncture collect blood collect one ml,

Procedure of ABO blood group;-

With a grease pencil two circle are drawn on clean and dry slide and labeled (A) and (B) on another slide a circle was drawn and labeled (D), and a drop of blood was placed on each circle then in circle (A) a drop of antisera D was added to circle D on the another slide. Then each suspension of each circle was mixed with different wooden stick (Lweis *et al.* ,1996).

2.7 Interpretation of ABO blood group:-

_ if Agglutination occurs on circle (A) and no agglutination occur on circle (B) the ABO blood group is A.

_ if Agglutination occurs on circle (B) and no agglutination occur on circle, (A) circle mean the ABO blood group is B.

_if Agglutination occurs on both (A) and (B) circles that the ABO blood group is AB.

_NO agglutination occurs on both (A) and (B) circle that the blood group is O.

_ if Agglutination occur circle on (D) the Rheuse D factor is positive.

_ if No agglutination occur on circle (D) the Rheuse D factor is negative.

2.8 Statistical analysis:-

All information about the study population was entered a computers as well as obtained result. The data was analyzed by using SPSS version 16 computer program was used. Frequencies and chi square test values were calculated.

Chapter three

Results

Total numbers of 150 diabetic patients were enrolled in the study during the period 17 March 2014 20 may2014.

Table (3.1) Distribution of study group according to gender and age

Type of DM	Gender		Age mean \pm SD Year
	male	female	
Type1	23(46%)	27(54%)	12.48 \pm 39.3
Type2	50(50%)	50(50%)	52.71 \pm 39.3
total	73	77	

Table 3.1 showed the majority of study group were females 77(51.3%) and 73(48.7%) were males.

46%of total study group were males with type 1 DM, 54% were females .

Both males and females with type2 DM are equal 50% .

Table (3.2) Distribution of age of type 1 diabetic patient

Age group (year)	Frequency	Percent %
2-10	12	74
11-20	37	24
21-30	1	2
Total	50	100%

Most common age group in type 1 DM ranged between 11-20 years, least age group ranged 21-30 years (table 3.2)

Table (3.3) Distribution of age of type 1 diabetic patient

Age group (year)	frequency	Percentage%
31-40	6	6
41-50	40	40
51-60	33	33
61-70	20	20
71-80	1	1
Total	100	100%

Show that most age group in type 2 DM ranged 41-50 year, least age group ranged 71-80 year(table 3.3)

Table (3.4) Distribution of study group according to duration of disease

Duration of disease	Type of DM			
	Type1 DM		Type2 DM	
	frequency	Percentage%	Frequency	Percentage%
1month-5year	27	54	50	50
6year-10year	17	34	33	33
11year-15year	6	12	11	11
16year-20year	0	0	3	3
21year-25year	0	0	3	3

Highest frequency of patient with diabetic duration between 1month and 5 year were noticed in patient with both types of DM.

Table (3.5) Distribution of blood group group according of males:-

Blood group		
	frequency	Percentage %
A +ve	19	26.4
A-ve	2	2.7
B +ve	20	27.3
B-ve	0	0
O +ve	22	30.1
O -ve	4	5.4
AB +ve	4	5.4
AB -ve	2	2.7

Table(3.5) show that most blood group in male is O +ve 22(30.1%) followed by B +ve 20(27.3%) then A+ve 19(26.4%).

Table (3.6) Distribution of blood group group according of females:-

Blood	Female
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group	Frequenc y	Percent age %
A +ve	17	22
A-ve	1	1
B +ve	14	19
B-ve	0	0
O +ve	37	48
O -ve	4	5
AB +ve	4	5
AB -ve	0	0

Table (3.6) most blood group in female is O+ve 37(48%) followed by A+ve 17(22%) then B+ve 14(19%)

Table(3.7) Distribution of study group according to blood group:-

			BLOOD GROUP								Total
			A+ve	A-ve	B+ve	B -ve	O +ve	O-ve	AB+ve	AB-ve	
type	type 1	Count	8	2	8	0	23	6	3	0	50
		% of Total	5.3%	1.3%	5.3%	0%	15.3%	4.0%	2.0%	.0%	33.3%
	Type 2	Count	28	1	26	0	36	2	5	2	100
		% of Total	18.7%	.7%	17.3%	0%	24.0%	1.3%	3.3%	1.3%	66.7%
Total		Count	36	3	34	0	59	8	8	2	150
		% of Total	24.0%	2.0%	22.7%	0%	39.3%	5.3%	5.3%	1.3%	100.0%

According to Table (3.6) most blood group in type1 DM is O +ve 23(15.3%), A+ve 8(5.3%),B+ve 8(5.3%),followed by O-ve 6(4%) ,then AB +ve 3(2%),A-ve 2(1.3%).

Most blood group in type 2 O+ve 36(24%),B+ve 26(17.3%),A+ve 28(18.7%),AB+ve 5(3.3%),O-ve 2(1.3%), AB-ve 2(1.3%),A-ve 1(.7%).

Chapter four

Discussion ,conclusion and Recommendation

4.1 Discussion

This is a cross sectional descriptive study aimed to association ABO blood group system ,Rh and diabetes mellitus .

The result showed that female patient in type 1 DM was more frequent than male. Among patient with type 2diabetes mellitus male and female were equal in number.

Highest age frequency of patient with type 1 diabetes mellitus was less than 20 years, but it was less than 50 years among patient with type 2 DM.

most duration of disease is between 1month-5year in both type 1 and type 2 DM that mean the incidence of diseases is increase

Regarding the association between ABO blood group system, the present study showed that blood group O +ve was most frequent among patient with both types of diabetes mellitus followed by A+ ve and B +ve , B -ve and AB -ve was least blood group.

Previous studies showed different results. A study in India by Sharma (2013) showed no association between ABO blood group and both types of DM (1 and 2) and B is most frequent blood group which disagree with the results of the present study, AB in the study of Sharma *et al.*,(2014) was least common blood group which disagree with result of current study while least blood group is B -ve in both types of DM.

Jassim (2012) in Iraq reported finding similar to the result of the present study, that O the blood group is the most frequent group among diabetic patients, but Jassim reported that blood group AB is least common which disagree with the result of current study.

Negative association between A and O blood group was reported by Kamil *et al.*, (2010) in Malaysia and no association between DM and blood group B and AB, which disagree with current study, least blood group is AB which disagree to current study.

A study in Pakistan by Waseem *et al* (2012) concluded that higher percentage in the diabetic group was AB (14.92%) compared with control, and blood group O has the same distribution among diabetic patient and control. The other conclude that there was no association between Rh and diabetes, but there was a negative association between blood group A and B.

The present result concluded that blood group O was the most frequent among patient with both types of DM and B-ve was the least frequent blood group.

Rh +ve was more frequent among diabetic patient.

4.2 Conclusion:

- Most blood group in diabetic patient in type 1 and 2 is blood group O with Rh positive factor.
- Most common age group in type 1 DM are ranged between 11-20 year , and most age group in type 2 DM are ranged between 41-50 year.
- The majority of study group were females and were males in type 1 DM.
- Male and Female patient in the type 2 DM showed same frequency.
- most duration of disease is between 1month-5year in both type 1 and type 2 DM that indicate increase incidence of the disease.

4.3 Recommendation:-

- The Rh blood types and subgroup need further investigation.
- Further research is highly recommended to determine the genetic and molecular variation that link ABO blood group and frequency of DM.

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**Sudan University of Science and
Technology**

Collage of Higher Studies

Medical laboratories Sciences

Hematology Department

**Questionnaires about ABO Blood Grouping in
Diabetic Patient**

Sample No ()

- Sex
- Age

- Type of
- Diabetes.....
- Duration of
- diseases.....
- Other.....

Lab Investigation:

- **Blood group**.....
- **Rh** (D)
- factor**.....

استمارة موافقة الشخص المشارك في البحث

أنا الباحثة دلال عبده محمد حسن **اقوم ببحث عن** علاقة الفصائل الدموية والعامل الريصي بمرض السكري في السودان

ونود ان نشير الي مشاركتك في البحث طوعية وان رفضك للمشاركة لاتفقدك الحق في اي فوائد من البحث , مع التاكيد علي انه لن يتم منحك اي قيمة نقدية مقابل المشاركة في البحث , كما نؤكد لك امكانية الانسحاب من البحث في وقت تشاء , ودون ابداء توضيح لأسباب الانسحاب , ولن يؤثر ذلك علي حقك في الاستفادة من البحث . اذا كان لديك اي سؤال او استفسار يخص البحث يمكنك الاتصال ع الرقم

فورم اقرار المشاركة في البحث

اقرار المشارك

لقد اطلعت علي المعلومات الحالية والتي تم شرحها لي واتيح لي طرح الاسئلة عنها كيفما شئت , ولقد تلقيت الاجابة الوافية عن كل الاسئلة وانا اقر بالموافقة علي المشاركة طواعية في هذه الدراسة واعلم بحقي في التوقف . عن المشاركة في وقت دون ان يؤثر ذلك علي حقوقي

..... أسم المشارك

..... توقيع المشارك

..... التاريخ

Quality control of reagent

Reagent

Anti-Human Globulin Anti-IgG, -C3d; Polyspecific is a blend of rabbit anti-IgG and murine monoclonal anti-complement (murine IgM AntiC3d, Bric 8). The anti-IgG component contains antibody reactivity against light IgG chains and thus may also agglutinate IgA or IgM coated red blood cells. The anti-complement component consists of murine monoclonal IgM anti-C3d-antibody reactive with C3b- and C3dcoated red blood cells. Antibodies are diluted in a isotonic saline

solution containing bovine albumin and as colorant Patent Blue and Tartrazin.

The following antibodies are produced using intermediate products produced for Biotest Medical Diagnostics GmbH in a shared manufacturing agreement with Millipore (UK) Ltd., 9 Fleming Road, Kirkton Campus, EH547BN, Livingston, UK; License Number 1721.

Anti-C3d clone BRIC 8 (IgM)

Preservative: 0.1% sodium azide.

Precautions

- For In-vitro diagnostic
- Store at 2 to 8°C.
- Do not use beyond the expiration date.
- Do not use if turbid.
- Do not dilute.
- Do not use specimens collected with gel separators.
- Handle and dispose of reagents as potentially infectious
- Caution: Do not pipette by mouth. The absence of all viruses has not been determined.
- Caution: This product contains Natural Rubber Latex Which May Cause Allergic Reactions.
- Warning: Contains sodium azide (NaN_3), which may react with lead or copper plumbing to form explosive azides. If discarded in the sink, flush with large amounts of water to prevent the build-up of explosive metal azides.
- The bovine albumin used for the production of this reagent is purchased from BSE-free US sources, Boval Company L.P. in Cleburne, Tx, USA and Millipore in Kankakee, IL, USA.

