Introduction

Hepatitis C virus (HCV) infections is a major worldwide public health problem with the highest prevalence in the world , ranging from 6%-28% with average 13.8% in the general population. The World Health Oanization (WHO) estimates that 3% of the worlds populations are chronically infected with HCV, most of these cases occur in Africa , which is reported to have the highest HCV prevalence rate . Although, direct percutaneous inoculation is the most efficient mode of transmission of HCV , several studies have demonstrated that sexual , household , occupational , and vertical transmission of HCV may also be of importance (ELShiekh *et al*.,2007).

Diabetes mellitus (DM) is a metabolic syndrome of multiple etiologies characterized by chronic hyperglycemia with disturbances of carbohydrates , fat and protein metabolism resulting from defects in insulin secretion , insulin action or both. DM may result from the distruction of the beta cells of the pancreas with consequent insulin deficiency or resistance to insulin action at the receptor site . DM can be broadly classified into type IDM , type 2 DM , gestational DM , and other specific types of DM (Olokoba *et al* .,2007).

Type 2 diabetes has become more prevalent as people become obese and live a more sedentary lifestyle . Risk factors strongly associated with type 2 diabetese include family history , body fat distribution , age , sex , smoking , and physical activity . It has also been suggested that in addition to these genetic , biologic , and demographic factors , HCV infection is associated with type 2 diabetes (Tuomilehto *et al* ., 2001).

A strong association between HCV and type 2 diabetes , may be explained by the association of the type 2 diabetes with HCV due to pathophysiology of HCV – associated type 2DM consists of a defect in insulin secretion , excessive hepatic glucose production , increased hepatic tumor necrosis

factor alpha , and insulin resistance . Emerging evidence in animals and humans has shown that HCV infection induces hepatic steatosis and increases tumor necrosis factor –a level , both resulting in the development of insulin resistance and subsequent type 2 diabetes (Hiroshi and Philip, 2006).

Most epidemiological studies have suggested that hepatitis C virus (HCV) infection is a rick factor for development of diabetes mellitus (DM) type2. The etological factors were initially thought to be cirrhosis but further studies differentiating between HCV and hepatitis B virus (HBV)related infection have shown that patients with HCV infection have a higher prevalence of diabetes mellitus type 2 (Mehta *et al* .,2003).

HCV is most efficiently transmitted through transfusion of infected blood transplantation of infected organs , and sharing injection drug equipment(Alter,1997).

Rationale:

Hepatitis C virus causes chronic liver disease . it is the major cause of liver fibrosis and lead to development of hepatocellular carcinoma .

World Health organization (WHO) estimated that 3% of word population chronically infected with hepatitis C virus most of these cases occur in Africa . This study was carried out to determine the prevalence of HCV infection a mong diabetic patients and to clarify the presence of any possible relationship between HCV and type 2 DM in this region

1.1 Objectives

1.1.1 General objective

To determine the frequency of HCV among patients with diabetes type 2.

1.1.2 Specific objective

1. To detect the presence of anti- HCV antibodies in diabetic patients.
2. To determine the rate of co-infection with both HCV Abs and other DM type2.
3. To to correlate between the presence of anti HCV Abs and other factors like gender , age , history of diabetes and smoking.

2.LITERATURE REVIEW

2..1 Hepatitis C Virus

Hepatitis C virus (HCV) is a positive strand RNA virus and the only member of the genus hepacivirus within the flavivirus family (Alter *et al.*,1992).

HCV was first identified in 1989 by researchers at the Centers for Disease Control and Prevention (CDC) in the United states ,when it was determined to be the primary cause of non-B hepatitis (Ballester *et al* ., 2005).

2.1.1. Genotypes

These are 6 genotypes of HCV , 52 subtypes within these genotypes , as well as diverse population of quasispecies within each infected individual . the source of this variation , like that of other RNA viruses , is the high mutation rate of its error prone RNA polymerase (Timm and Roggendorf.,2007).

2.1.2 Classification of HCV

HCV is the only known member of the hepatitis virus genus in the family flaviridae .there are six major genotypes of HCV, which are indicated numerically (1-6) with several subtypes within each genotypes to represent letters subtypes are further broken down quasi species based on their genatic diversity (Beek and Dubussion ,2003).

2.1.3 virus structure and replication

The genome is approximately 9.6 Kb and encodes an approximately 3000 aminoacidpolyprotien . It is flanked by 50 and 30 untranslated regions (UTRs), that are required for replication and the initiation of translation .

The 50 UTR contains extensive secondary structure such as an internal ribosome entry site (IRES) that directs translation ,binds to ribosomal protein (Buratti *et al*.,1998).

In addition , other sequences in the 50 UTR are required for replication of te negative strand .Aliver specific micro RNA ,miRNA-122,with binding sites in the 50 UTR has been shown to facilitate HCV replication (Jopling *et al*.,2006).The 30 UTR also contains extensive secondary structure and is required for replication .

There is evidence for long-range RNA/RNA interactions between the 50 and 30 UTRs as well as between the 50 UTR andRNA sequences at the C-terminus of NS5B .

These interactions are essential fir replication and strongly enhance translation from the HCV IRES(5-7).the 50 and 30 UTRs may also be required for encapsidation as they both interact with the core protein (Yu *et al*.,2009) . After translation on the rough endoplasmic reticulum (ER), the polyprotien precursor is cleaved into 10 proteins by a combination of host and viral protease .

These protein then associate with ER and modify cellular membranes producing the memberanous web upon which viral replication occurs (Yu *et al*., 2009)and (Lai *et al* .,2008).

Virus mutation and assembly occurs in association with lipid droplets and appears to hijack the VLDL secretion machinery for viral egress from the cell(Gastaminza *et al* .,2008).

The P7 protein is a viroporin that may perform a function similar to the M2 protein of influenza , it is dispensable for replication but essential for assembly and release of infectious virions (Brohm,*et al*.,2009).

The structure protiens ,core,E1,E2,and the viral porin p7 are processed by host signal peptidase (Hijikata *et al* .,1991) .the core protein is further processed by host signal peptidase to yield its mature form that can associate with lipid droplets (Yu *et al*.,2009).

The core protein which forms the viral capsid, has been shown to interact with both the 50 and 30 U TRs (8, as well as with the envelope glycoprotein)E1 this interaction is depending on oligomerization of core (Nakai *et al*.,2006).

In addition, since mutations in core can be rescued by compensatory mutations in NS2 and p7,it has been proposed that they too interact with core (Muray *et al*,.2007)

The two envelope glycoproteins form heterodimers on the ER(Deleersmyder *et al*,.1997)and are glycosylated there, but not further modified by Golgi enzymes indicating that they are retained in the ER(De Beeck *et al*.,2004)

2.1.4 Stability

HCV is inactivated by exposure to lipid solvents or detergents heating at 60C for 10 hrs or 100c for 2 min in aqueous solution ,formaldehyde (1:2000)at 37Cfor 72 hrs B"-propriolactone and UV irradiation.

HCV is relatively unstable to storage at room temperature and repeated freezing and thawing (WHO,2013).

2.1.5 pathogenesis induced by HCV:-

Systemic (omics) approaches are beginning to unravel the host cell networks that are involved in HCV pathogenesis it has long been known that HCV infections in patients leads to apoptosis , which stimulates both Kuppffer and stellate cells to produce TGF –b leading to activation of stellate cells and the deposition of collagen .

These repeated cycles of liver damage (apoptosis) and collagen deposition eventually leads to fibrosis ,cirrhosis and liver failure . The apoptosis of hepatocytes was thought to be mediated by the adaptive immune response to HCV infected cells (Nelson,1998).

However, two recent papers pointed to agreater role for the hepatocyte in both apoptosis and fibrosis . It was shown that apoptosis is induced by HCV

infection in mice with chimeric mouse/human livers lacking an adaptive immune system (Joyce,*et al*.2009).

Moreover,the apoptosis is specific to HCV infected cells .this effect was mediated by a combination of induction of ER and oxidative stress and the down regulation of anti-apoptotic proteins NF-KB and Bcl-xl in infected hepatocytes .

It was hypothesized that the initial apoptotic signal came from Kupffer or Natural killer cells, and that this is the initial step in liver damage which precedes the activation of stellate cells and fibrosis . Using transcriptional profiling of infected Huh7.5 cells ,it has been shown that in addition to induction of oxidative stress , and apoptosis markers , genes associated with TGF-b signaling were induced , in the absence of other cells types (Walters *et al*.,2009).

2.1.6 diagnosis

This disease is often a precursor to potentially fatal disease , such as cirrhosis and hepatocellular carcinoma . Consequently , the United States National Institutes of Health (NIH) recommends testing for hepatitis C in humans with a history of transfusion of blood or blood-products before 1990 ,on hemodialysis , who have had multiple sexual partners , who are spouses or household members of HCV patients , or who inject drugs or share instruments for intranasal drug administration. Testing is important because humans with hepatitsC are typically asymptomatic or have only minor , nonspecific symptoms .(Astruthers Ph.D university of the sciences in Philadelphia ,2007).

Diagnosis of hepatitis is made by biochemical assessment of liver function .

Initial laboratory evalution should include:total and direct bilirubin , ALT , AST , Alkaline phosphatase , prothrombin time , total protein , albumin ,globulin , complete blood count , and coagulation studies (Houghton, 1996).

The first recommended test is sensitive and specific , is reproducible and inexpensive , and thus is appropriate for screening at risk populations . HCV

RNA assays may be used to confirm the diagnosis . Testing for serum ALT is inexpensive and non invasive but is less sensitive for determining disease status . A problem is that about 30% of patients with chronic HCV have normal ALT . Repeated ALT testing over time may allow a better assessment of liver injury , but this has not been clearly documented . Aliver biopsy cannot serve to diagnose HCV infection , although it can provide useful histological information on liver injury . According to the NIH consensus statement (Astruthers Ph.D University of the Sciences in Philadelphia , 2007).

2.1.7 treatment

The rationale for treatment of chronic hepatitis are to reduce inflammation , to prevent progression to fibrosis , cirrhosis , and hepatocellular carcinoma (HCC) through the eradication of the virus in chronically infected patients ,and to decrease infectivity and control the spread of the disease (Who ,2003) .

Treatment for chronic hepatitis C should start when patients have 3 indicators of HCV :

*abnormal ALT for over 6 months .

*Positive HCV RNA

*liver biopsy has shown fibrosis and signs of necrosis and inflammation .

In chronic hepatitis C , the anti-viral effect of alpha interferon is well demonstrated, with arapid decrease of serum HCV RNA within the first weeks of therapy , with a parallel decrease of serum ALT (Hoofnagle and Bisceglie , 1997).

Ribavirin is a guanosine –like nucleoside analog which has abroad spectrum of antiviral activity against several viruses (Reichardet *et al.*,1991) .

Combination therapy results in better treatment responses than monotherapy ; the highest response rates have been achieved with pegylated interferon in combination with ribavirin .Interferon has been shown to normalize liver tests , improve hepatic inflammation and reduce viral replication in chronic hepatitis C and considered the standard therapy for chronic hepatitis C (WHO ,2003).

2.2Diabetes mellitus (DM)

2.2.1 Definition

The term diabetes mellitus describes a metabolic disorder of multiple a etiology characterized by chronic hyperglycemia with disturbances of carbohydrate ,fatand protein metabolism resulting from defects in insulin secretion , insulin action , or both . The effects of diabetes mellitus including long-term damage, dysfunction and failure of various organs .

Diabetes mellitus may present with characteristic symptoms such as thirst , polyuria , blurring of vision, and weight loss . In its most severe forms , ketoacidosis or a non-ketotic hyperosmolar state may develop and lead to stupor , coma and , in absence of effective treatment , death. Often symptoms are not severe , or may be absent , and consequently hyperglycaemia sufficient to cause pathological and functional changes may be present for a long time before the diagnosis is made . The long – time effects of diabetes mellitus include progressive development of the specific complications of retinopathy with potential blindness , nephropathy that may lead to renal failure , and/or neuropathy with rick of foot ulcers , amputation, charcoal joints , and features of autonomic dysfunction, including sexual dysfunction .

People with diabetes are at increased risk of cardiovascular , peripheral vascular and cerebrovascular disease.

Several pathogenic processes are involved in the development of diabetes.

These include processes which destroy the beta cells of the pancreas with consequent insulin deficiency , and others , that result in resistance to insulin action. The abnormalities of carbohydrate, fat and protein metabolism are due to deficient action of insulin on target tissues resulting from in sensitivity or lack of insulin (WHO.1999).

2.2.2 Main Types of Diabetes

The three main types of diabetes are type -1 , type -2 and gestational diabetes :

- Type 1 diabetes , formerly called juvenile diabetes , it usually first diagnosed in children , teenagers , and young adults . In this type of diabetes , the beta cells of the pancreas no longer make insulin because the boys immune system has attacked and destroyed them .
- Type 2 diabetes, formerly called adult –onset diabetes , isthe most common type of diabetes . about 90 to 95 present of people with diabetes have type 2.

  People can develop type 2 diabetes at any age , even during childhood , but this type of diabetes is most often associated with older age . Type 2 diabetes is also associated with excess weight , physical inactivity , family history of diabetes , previous history of gestational diabetes , and certain ethnicities (National Diabetes Statistics Report , 2014).
- Type 2 diabetes usually begins with insulin resistance , a condition linked to excess weight in which muscle , liver, and fat cells do not use insulin properly . As a result , the body needs more insulin to help glucose enter cells to be used for energy .At first , the pancreas keeps up with added demand by producing more insulin . But in time , the pancreas loses its ability to produce enough insulin in response to meals , and blood glucose level rise (National Diabetes Statistics Report , 2014).
- Gestational diabetes is a type of diabetes that develops only during pregnancy . The hormones produced during pregnancy increase the amount of insulin needed to control blood glucose levels .If the body can not meet this increased need for insulin , women can develop gestational diabetes during the late stages of pregnancy . Gestational diabetes usually goes away after the baby is born. Shortly after

pregnancy , 5 to 10 present of women with gestational diabetes continue to have high blood glucose level and are diagnosed as having diabetes , usually type 2 (National Diabetes Statistics Reports ,2014)

2.2.3 Symptoms of Types 2 Diabetes

The symptoms of type 2 diabetes typically develop slowly over time (that is, it is a chronic rather than an acute condition) . some people with mild type 2 diabetes have no symptoms and may be unaware that they have the condition . Symptoms of diabetes may include increased hunger and thirst , frequent urination, blurred vision, itchy skin (Liz ,2001) .

2.3 Previous studies

There are many studies which indicate a high prevalence of HCV infection among diabetic patients .In sudan prevalence rate of 1.7% for HCV infection was recorded among type 2 DM patients with no seropositivity detected among the control group of volunteer blood donors without diabetes (Ahmed and Adam , 2014).

In India , out of 192 type 2 DM patients screened , prevalence rate of HCV sero-positivity is found to be 11/192(5.7%) (Demitrost ,2015).

An Egyptian study showed that

incidence of T2DM increased two fold in patients who had HCV infection compared with those who did not and reported that HCV infected persons with diabetes mellitus were T2DM more likely to need insulin (EL – Zayadi *et al* ., 1998).

In Pakistan in study found 36% from 100 DMT2 patients , (Ali *et al*., 2007).

In Nigeria study showed that the prevalence rate of HCV infection was 33(11%) . In response to diabetic status , females subjects had a higher prevalence of 178 (59.3 %) compared to males 122(40.7%) (Ndako *et al* .,2009).

In America it was found a higher prevalence of HCV seropositivily in DM patients than in the general population (Bahtiyar *et al* .,2004).

Study on Yemeni patients found that in type 2 diabetes patients 7 out of 50 (14%) detected with hepatitis , 5 (10%) of 50 type 2 diabetes patients had evidence of HCV infection compared to 2 (4%) with HBV . The development diabetic mellitus among 70 hepatitis C and B patients 8 out of 70 (11.4%), 3 HBV 36 (8.3%) , 4 HCV 29 (13.7%) . Co- infection HBV and HCV 5 (20%) compared to 1 (2%) without association in 50 control adults (Habib *et al* .,2014).

3. MATERIALS AND METHODS

3.1 Study design

3.1.1Type of study

This was an analytical , descriptive study conducted in the period from March to July 2017

3.1.2 Study area

This sudy was groups carried out in Marawi al skary Hospital in Northern State

3.2 Inclusion criteria

Confirmed Type 2DM with age ranged from 35-65 years included In three age groups (35-45years, 46-55 years and 56-65years)

3.3 Exclusion criteria

Subjects were type 1 diabetes , transplant recipients , emergency cases and dialysis patients were excluded.

3.4 Ethical consideration

Approval to conduct this study was obtained from college Ethical committee , Sudan university of Science and Technology .Verbal concent was taken from the participants after informed by the purpose of the study.

3.5 Sample size and Sampling Technique

Non –probability sampling method was used (only those who voluntereered were involved in sample collection)

Atotal of ninety (n=90) patients were involved in this study

3.6 Tool of data collection

Astructured questionnaire was used for collection of data which includes demographic information such as age, material status , education level ,

family history of HCV infection , previous surgery or jaundice , previous needle stick , and sharing of some article such as razor and nail clip

3.7 Laboratory method

3.7.1 Serum specimens collection

The blood specimens were collected using the venipuncture for collection . The suitable vein was located , using sterile syringe (5ml)to collect the blood after cleaning the skin area with alcohol pads and The blood was dispensed in a sterile plain container .

3.7.2 Preparation of the specimens

Blood specimens after clotting , were centrifuged at 3000 round/minute for 5 minutes to obtain serum, and then the obtained sera were collected in clean sterile containers properly labeled and kept at -20C till used .

3.8 ELISA for detection of HCV

3.8.1 Principle of the assay

This is an ELISA for qualitative detection of antibodies against Hepatitis C virus in human serum or plasma .it is intend for screening blood donors and diagnosing patients with HCV .

It employs a solid phase ,indirect ELISA method for detection of antibodies to HCV in two steps incubation procedure , polystyrene micro well strips are precoated with recombinant , highly immunoreactive antigen (Ags) corresponding to the core and the non-structural regions of HCV (Third generation HCV ELISA) . during the first incubation step, anti HCV specific antibodies , if present ,will be bound with HCV Ags pre-coated solid phase .

The well are washed to remove un bound serum proteins , and rabbit anti -human IgG antibodies (anti-IgG) conjugated to horse reddish peroxidase (HRP-conjugated) are added .during the second incubation step, these HRP-conjugated antibodies will be bound to any antigen-antibodies

(IgG)complexs previously formed and the un bound HRP-conjugate is then removing by washing . chromogen solutions containing tetramethylbenzidine (TMB) and urea peroxide eare added to the wells and in present of antigen – antibody – anti –IgG (HRP) immune complex ; the color less chromogens are hydrolyzed by bound HRP conjugate to blue- color product . the blue color turns yellow after stopping the reaction with sulphuric acid . the amount of color intensity can be measured and in a proportional to the antibody captured in the wells , and to the sample respectively . wells containing samples negative for anti-HCV remain colorless .

3.8.2 Assay procedure

Step 1- reagent preparation

The reagens and samples were allowed to reach room temperature (18-30^{o}**C**)for 15 minutes .The stock wash buffer had been diluted 1 to 20 with distilled water.

Step 2- numbering the wells

Three wells were marked as negative control (B1, C1 , D1), two wells as positive control (E1 , F1) and one blank (A1).

Step 3- adding diluent

100Ml of diluent were added into each lwell except the blank.

Step 4- adding samples

10Ml of negative controls , positive controls and samples were added into their respective wells.

Step 5- incubation (1)

The plate was covered and incubated for 30 minutes at 37 ºC.

Step 6- washing (1)

The plate cover was removed and discarded .The wells were washed 5 times with diluted wash buffer(Tween 20) . Each time ,allowing the

micro wells to soak for 30-60 seconds , after the final washing cycle the strips plate was turned onto blotting paper or clean towel , and taped to remove any remainders .

Step 7- adding HRP- conjugate

Hundred µL of HRP –conjugate were added in to each well except the blank .

Step 8- incubation (2)

The plate was covered and incubated for 30minutes at 37 ºC.

Step 9- washing (2)

The plate was removed and discarded .The wells were washed 5 times with diluted wash buffer as in step (6) .

Step 10- colouring

Then 50µl of chromogen A And 50µl of chromogen B solution was dispensed into each well including the blank and mixed by tapping the plate gently and incubated for 15 minutes at 37 ºC avoiding light .

Step 11- stopping the reaction

Using multichannel pipette 50µl stop solution (H_2SO_4)was added into each well and mixed by tapping the plate gently .

Step 12- measuring the absorbance

The plate reader was calibrated with the blank well and read the absorbance at 540 nm , and calculated the cut-off value and evaluated the result .

3.8.3 Calculation QC ranges

Calculation of cut off

Cut-off (c.o.) = mean NC +0.12

Quality control ranges

The value of the blank well should be less than 0.08 at 450nm.

3.9 Interpretation

Negative results

Samples giving an absorbance less than cut off value were considered negative which indicated that no antibodies to hepatitis C virus had been detected .

Therefore the patient was probably not infected .

Positive results

Sample giving an absorbance equal to or greater than cut off value were considered initially reactive , which indicates that antibodies to hepatitis C virus had been detected .

3.10 Data analysis

The data that collected from questionnaire and laboratory results were analyzed using the computer program SPSS version 20 and Chi –Square test was used .

4. Results

This study was carried out during the period from March to July 2017 including 90 diabetic patients with age range from 35-65 years .

From 90 diabetic patients , 1 (1.1%) was found to have anti- HCV antibodies within age group 56--65 years , table (1) shows frequency of anti -HCVamong age groups above 35 years and gender with positive results ,one were male and frequency of anti –HCV and age groups seen in table (2) .

Table (1) : frequency of anti –HCV and gender

Gender	No.of patients	No .of anti-HCV Positive	Percentage of Positive
Males	48	1	1.1%
Females	42	0	0%
Total	90	1	1.1%

Table (2) : frequency of anti –HCV and age groups

Age groups	No.of patients	No.of anti-HCV positive	Perecentage of Positive
35-45	15	0	0%
46-55	35	0	0%
56-65	40	1	1.1%
Total	90	1	1.1%

Table (3) shows correlation between the presence of HCV antibodies (Abs) with smoking , and only one patient was smoker.

Table (3) : correlation between the presence of HCV Abs and smoking

Smoking	**HCV Results positive**		**HCV Resulte negative**		**Total**
	Frequency	**Percent**	**Frequency**	**Percent**	
Smoker	1	1.1%	14	15.6%	15
Non smoker	0	0%	75	83.3%	75
Total	1	1.1%	89	98.9%	90(100%)

Chi-square test P value(0.025)

Table (4) shows the correlation between the presence of HCV Abs and family history of DM , it was found that one was having family history to the diabetes.

Table (4) :correlation between the presence of HCV Abs and family history of DM

History of diabetes	HCV positive Results		HCV negative Resulte		Total
	Frequency	Percent	Frequency	Percent	
History	1	1.1%	54	60%	55
No history	0	0%	35	38.9%	35
Total	1	1.1%	89	98.9%	90(100%)

Chi-square test P value(0.422)

5. Discussion

5.1 Discussion

HCV infection is a major worldwide public health problem and they have great affect on community and future generations health . So there is a need to conduct studies that may give guidelines for proper planning to deal with the health problem that related to HCV infection .

The present study aimed at detection of HCV among diabetic patients in Northern State . Out of 90 blood specimens investigated only 1 (1.1%) were positive the remaining 89 (98.9%)were negative .

This result is similar to that obtained in Blue Nile State , Sudan by Ahmed and Adam (2014). A case control study was conducted to determine frequency of HCV among diabetic patients type 2 using ELISA fourth generation for anti-HCV antibodies was done in 180 samples of patients with type 2 DM visiting El-Roseires Hospital , and 180 volunteer blood donors visiting blood bank of the same hospital ,reported thate it was (1.7%) .

The main difference in results is obviously due to the large sample size (180) they included and patients may be under treatment at the time of study .

But the result was lower than other studies . It disagrees with that reported in Nigeria out of three hundred (300) confirmed type 2 diabetic patients were screened for hepatitis C virus antibodies at the plateau State Specialist Hospital Jos , using grand diagnostic test strip , study showed that the prevalence rate of HCV infection was 33 (11%) and patients with out family history of diabetes showed a higher seroprevalence of 13 (6.7%) (Ndako *et al* .,2009) .

In this study it was observed that older patients were more likely to have HCV infection as compared to those in the younger age groups .

Difference in results may be due to epidemiology of the disease , personal hygiene , different educational level and a wareness about disease transmission .

5.2 Conclusion

This study showed that frequency of HCV amonnge diabetic patient type 2 was 1/90 (1.1) within age group 56-65 years , that was male .

There was asignificant association between HCV infection and type 2 diabetes in the region according to the findings of the present study .

Also evident that certain factors including older age , male gender, long duration of diabetes significantly increased the rick of having HCV infection .

All the study group had neither previous blood transfusion nor alcohol intake.

5.3 Recommendation

1. increase the educational level about the virus ,its transmission and prevention for diabetic patients.
2. diabetic patients should be screened regularly for HCV to control and minimize development of complication .
3. fruther studies with large number of samples and more advanced technique are required to validate the results of the present study .

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