1. INTRODUCTION

1.1 Introduction

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 destruction 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 1DM, type 2 DM, gestational DM, and other specific types of DM (Olokoba*et al.*, 2007).

There is an increasing prevalence of diabetes mellitus (DM) worldwide. It has been projected that 300 million people worldwide will have the condition by 2015. Type-2 DM constitutes about 90% of the entire population with DM; hence type-2 DM will form the bulk of the estimated increase in prevalence by the year 2015 (Amos *et al.*, 2010).

Type 2 diabetes has become more prevalent as people become more obese and live a more sedentary lifestyle. Risk factors strongly associated with type 2 diabetes 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).

The awareness of viral hepatitis has increased over the past few decades. Hepatitis C virus (HCV) infection is a common cause of acute and chronic hepatitis, and leads to cirrhosis and hepatocellular carcinoma. It is estimated that nearly 150 to 200 million people have been in contact with HCV worldwide, and approximately 85% have chronic infection. Several studies

from different parts of the world have found that 13% to 33% of patients with chronic HCV have associated diabetes, mostly type 2 DM (Chehadeh*et al.*, 2009).

Type 2 DM accounts for over 90% of all cases of DM, over 100 million people are diabetic worldwide and probably as many are not diagnosed (Alberti*et al.*, 1990).

Hepatitis C virus (HCV) infection is an important public health problem which currently affects more than 170 million people (about 3% of world population) out of which 55-80% have chronic infection (NIH, 2002).

Infection with HCV has been shown to produce both hepatic and extra hepatic manifestations, the latter include insulin resistance, essential mixed cryoglobulinemia, glomerulonephritis, porphyria cutaneous tarda and benign monoclonal gammopathy (Zignego*et al.*, 2007).

A meta-analysis showed that HCV increases the risk of Type 2DM by 1.8 times in excess of that posed by relative degree of liver pathology (White *et al.*, 2008).

Astrong association between HCV and type2 diabetes, may be explain the association of 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).

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

Most persons who become infected with HCV viremia persist, accompanied by variable degrees of hepatic inflammation and fibrosis. Earlier studies of chronic HCV infection suggests that only a small number of hepatocytes become infected, but more recent studies suggest that 50% or more harbor the virus (Agnello*et al.*, 1999).

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

Rationale:

HCV infections are common worldwide. It is estimated that about 3% of the world's population have HCV (WHO, 2003).

In Sudan, HCV was detected in pregnant women, schistosomic school children and found to be the major cause of transfusion associated non-A non-B hepatitis, 50% of infected individuals develop severe life threatening chronic hepatitis with liver cirrhosis and hepatocellular carcinoma.

Also, Ahmed and Adam(2014) found statistically significant association between HCV and type 2 DM (1.7%).

A large number of clues have suggested the potential role of a common hepatotrophic virus in developing diabetes. New observational studies in which prevalence of HCV infection in patients with DM have been carried out in endemic countries. This study was, therefore, carried out to determine the prevalence of HCV infection among diabetic patients and to elucidate the presence of any possible relationship between HCV and Type 2DM in this region.

1.1 Objectives

1.1.1 General objective

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

1.1.2 Specific objectives

1. To detect the presence of anti-HCV antibodies in diabetic patients.
2. To determine the rate of co-infection with both HCV and DM type 2.
3. 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 flavivirus family (Alter *et al*., 1992).

Hepatitis C virus was first recognized as a separate disease entity in 1975 when the majority of transfusion related hepatitis were found not to be caused by the only two hepatitis viruses recognized at that time i.e. Hepatitis A virus and Hepatitis B virus. The disease at that time was called “non-A non-B hepatitis. The discovery of hepatitis C genome in 1989 has now lead to the realization that this virus is a major health problem worldwide (Purcell, 2007).

2.1.1 Structure of HCV Virion

HCV particle is small 55-65 nm in size, consists of core genetic material (RNA) surrounded by classical icosahedral scaffold protective shell of protein, and further encased in lipid envelope of cellular origin (Beek and Dubussion, 2003).

2.1.2 Classification of HCV

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

2.1.3 Stability

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

HCV is relatively unstable to storage at room temperature and repeated freezing and thawing (WHO, 2003).

2.1.4 Replication of Hepatitis CVirus

Involve several steps, the viruses need a certain environment to be able to replicate, and must therefore, first move to such area. HCV has high rate of replication with approximately one trillion particles produced each day in an infected individual. Due to lack of proof reading by HCV RNA polymerase as HCV also has exceptionally high mutation rate, a factor that may help it elude the host´s immune response (Linderbash*et al.*, 2005).

Once inside the hepatocytes, HCV mainly replicates within them. However there is controversial evidence for replication in lymphocyte or monocytes bymechanism of host tropism. Circulating HCV particles bind to receptors on the surface of hepatocytes and subsequently enter the cells. Two putative HCV receptors or CD8- 1 and human scavenger receptor classB1 (SB-B1) (Linderbash*et al.*, 2005).

However, these receptors are found throughout the body. The identification of hepatocyte-specific factors that determine observed HCV liver tropism is currently under investigation (Linderbash*et al.*, 2005).

Once inside the hepatocytes, HCV initiates the lytic cycle accomplish it is own replication. The polyprotein is then proteolytically processed by viral

and cellular protease to produce three structural (virion associated) and no seven structural protein (NS) (Linderbash*et al.*, 2005).

Alternatively, a frame shift may occur in core region to produce an alternate reaching from protein (ARFP) HCV include two proteases. The NS protein then recruits the viral genome into RNA replication complex, which is associated with rearranged cytoplasmic membranes (Linderbash*et al.*, 2005).

RNA replication takes place via the viral RNA dependent RNA polymerase NS5B, which produce strand RNA then serves as a template i.e. production of new positive strand viral genomes. Ascent genomes can then be tanslated, further replicated, or packaged within new virus particles, new virus particles are through to bud into secretary pathway and release at the cell surface (Linderbash*et al.*, 2005).

2.1.5 Diagnosis

Diagnosis of hepatitis is made by biochemical assessment of liver function. Initial laboratory evaluation should include: total and direct bilirubin, ALT, AST, alkaline phosphatase, prothrombin time, total protein, albumin, globulin, complete blood count, and coagulation studies (Houghton, 1996).

Hepatitis C diagnosis depends on demonstration of anti-HCV antibodies detected by an Enzyme Immune Assay (EIA). Anti-HCV is generally not detectable in patients with initial signs or symptoms of hepatitis C. Anti-HCV develop in acute infection generally between 2 and 8 weeks after evidence of liver injury. Some persons may not test positive for 6-9 months after onset of illness. Hepatitis C viremia may be detected by RT-PCR within days after infection (Marcellin, 1999).

Third generation enzyme linked immunosorbent assays (ELISA) and recombinant immunoblot assays (RIBA) have considerably improved in sensitivity and specificity as compared with prior first and second generation assays. Techniques for HCV-RNA detection and quantification, such as the branched DNA (bDNA) and polymerase chain reaction (PCR), are more standardized and the sensitivity has been improved (Pawlotsky*et al.*, 1998).

2.1.6 Treatment

The rationales 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).

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

Ribavirin is a guanosine-like nucleoside analog which has a broad spectrum of antiviral activity against several viruses (Reichard*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 is considered the standard therapy for chronic hepatitis C (WHO, 2003).

2.2 Diabetes mellitus (DM)

2.2.1 Definition

The term diabetes mellitus describes a metabolic disorder of multiple aetiology characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both. The effects of diabetes mellitus include 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-term 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 risk of foot ulcers, amputation, Charcot joints, and features of autonomic dysfunction, including sexual dysfunction. People with diabetes are at increased risk of cardiovascular, peripheral vascular and cerebrovascular disease.

Several pathogenetic 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 insensitivity 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, is 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 body's immune system has attacked and destroyed them.

• Type 2 diabetes, formerly called adult-onset diabetes, is the most common type of diabetes. About 90 to 95 percent of people with diabetes have type2.

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 the 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 levels 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't 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 percent of women with gestational diabetes continue to have high blood glucose levels and are diagnosed as having diabetes, usually type 2 (National Diabetes Statistics Report, 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, fatigue, blurred vision, and dry, itchy skin (Liz, 2001).

2.3 Possible Pathogenesis of HCV Infection in the Development of Type2 DM

The link between the HCV and diabetes was first reported by Allison *et al*. in 1994 and later explored by Simo and colleagues in 1996 (Simo *et al.*, 1996) .The initial idea that patients with T2DM have more parenteral exposures because of use of finger stick devices and thus are at an increased risk of contracting blood borne infections such as HCV was disproved by a study from France in1998 (Rudoni*et al.*, 1999).

The pathogenic mechanisms causing DM in patients with HCV infection are still not well understood, although both insulin resistance and impaired insulin secretion have been considered to play an important role in the development of DM. In a transgenic mouse model, the reduction of plasma

glucose concentration after intraperitoneal insulin injection was impaired in HCV core-gene transgenic mice, displaying a higher plasma glucose level than in control mice, as well as significantly higher basal serum insulin levels, indicating insulin resistance in HCV core-gene transgenic mice (Shintani*et al.*, 2004).

Several studies have demonstrated insulin resistance in patients with HCV-related chronic hepatitis, using a homeostasis model assessment (HOMA) demonstrated that, both insulin resistance and beta-cell dysfunction contributed to glucose intolerance in chronic hepatitis C patients (Narita *et al.*, 2004).

Delgado-Borrego *et al, (*2004) reported that HCV is independently associated with increased insulin resistance among orthotopic liver transplant recipients.

One possibility is that HCV may infect and damage the insulin-producing beta cells of the pancreas. Since HCV is known to be associated with autoimmune conditions, another possibility is that the immune systems of people with HCV may attack and damage insulin-producing cells. In addition, liver inflammation or damage due to HCV may affect the production of glucose or the metabolism of insulin by the liver, thus altering blood sugar levels. Studies have shown that cirrhosis of the liver, regardless of cause, increases the risk of insulin resistance, although it is not known why (Liz, 2001).

Petit *et al,* (2001) demonstrated that insulin resistance in non-diabetic HCV-infected patients is correlated with the staging of liver fibrosis and may occur early in the course of HCV infection, even in non-diabetic patients.

More recently, the role of tumor necrosis factor (TNF)-α in the pathogenesis of DM in chronic hepatitis C patients has gained extensive interest (Knobler*et al*., 2003).

TNF-α has been shown to inhibit insulin-stimulated tyrosine phosphorylation of insulin receptor and insulin receptor substrate 1 in adipocytes, stimulate lipolysis, and increase serum-free fatty acids, leading to insulin resistance in muscle and liver, mediate hepatic insulin resistance to increase hepatic glucose production, and down-regulate genes in adipocytes encoding proteins such as insulin receptor substrate 1, glucose transporter-4, peroxisome proliferator-activated receptors, and adiponectin. In addition, TNF-α may reduce beta-cell function by direct toxic effects, further contributing to the development of DM. Recent studies have shown significantly higher levels of soluble TNF- α receptors in diabetic HCV patients than in non-diabetic HCV patients and controls (Knobler and Schattner, 2005).

2.4 Previous studies

In Sudan prevalence rate of 1.7% for HCV infection was recorded among type2DM patients with no seropositivity detected among the control group of volunteer blood donors without diabetes (Ahmed and Adam, 2014).

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).

A large retrospective survey of 1332 Italian patients with cirrhosis found that type 2 diabetes mellitus was present in 23.6% of those with HCV infection and in 9.4% of those with HBV infection (Caronia *et al.*, 1999).

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).

Bahtiyar*et al* (2004) found a higher prevalence of HCV seropositivity in DM patients than in the general population in the United States of America.

Ali *et al* (2007) in their study amongst Pakistanis found that HCV infection occurred more in DM patients.

Similarly, in Faisalabad study show that out of 154 diabetic patients, 18.83% (n=29) were positive for anti HCV antibody. The highest prevalence of anti HCV antibody was seen in the age group 41-50 years (Yahya and Iqbal, 2011).

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

This was an analytical, descriptive study conducted in the period from February to May 2015.

3.2 Study area

This study was carried out in Zinam Diabetic Hospital at Khartoum City.

3.3 Ethical concideration

Ethical approval from the Ministry of Health and informed consent regarding data and blood samples were obtained for collection and examination of the samples.

3.4 Inclusion criteria

Confirmed Type 2DM with age ranged from (45years - 65years) included in two age groups (45-55 years and 56-65 years).

3.5 Exclusion criteria

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

3.6 Sampling technique

Non-probability sampling method was used (only those who volunteered were involved in sample collection).

3.7 Serum Specimens collection

Three ml of blood samples were obtained via vein puncture in plain tubes. The blood samples after complete clotting were centrifuged at 3000 round/minute for 5 minutes, Sera were then collected in clean sterile containers properly labeled and kept at -20 ◦C till used.

3.8 ELISA for detection of HCV

3.8.1 Principle of the assay

This kit employs solid phase, indirect ELISA method for detection of antibodies to HCV in two-step incubation procedure. Polystyrene microwell strips are pre-coated with recombinant, highly immunoreactive antigens corresponding to the core and non-structural regions of HCV (Fourth generation HCV ELISA)(Fortress Diagnostics Limited, United Kingdom). During the first incubation step, anti-HCV specific antibodies, if present, will be bound to the solid phase pre-coated HCV antigens.

The wells were washed to remove unbound serum proteins and rabbit anti-human IgG antibodies (anti- IgG) conjugated to horseradish peroxidase (HRP-Conjugate) is added. During the second incubation step, these HRP-conjugated antibodies will be bound to any antigen-antibody (IgG) complexes previously formed and the unbound HRP-conjugate was then removed by washing.

Chromogen solutions containing Tetramethylbenzidine (TMB) and urea peroxide were added to the wells and in presence of the antigen-antiboy-

anti-IgG (HRP) immunocomplex; the colorless Chromogens are hydrolyzed by the bound HRP conjugate to a blue-colour product. The blue colour turns yellow after stopping the reaction with sulphuric acid. The amount of colour intensity can be measured and is proportional to amount of antibody captured in the wells, and to the sample respectively. Wells containing samples negative for anti-HCV remain colourless.

3.8.2 Assay procedure

Step 1- Reagents preparation:

The reagent and samples were allowed to reach room temperature (18-30℃) for at least 15-30 minutes. Then checked the wash buffer concentrate for the presence of salt crystals. If crystals have formed in the solution, re-solubilized by warming at 37℃ until crystal dissolved. The stock wash buffer was diluted 1 to 20 with distilled water.

Step 2- Numbering Wells:

The strips needed were set in strip-holder and numbered the wells including three negative control (B1, C1 and D1), two positive control (E1, F1) and one blank (A1).

Step 3- Adding Diluent:

Then 100µl of specimen diluent was added into each well except the blank.

Step 4- Adding Sample:

Then 10µl of positive control, negative control and specimen was added into their respective wells and mixed by tapping the plate gently.

Step 5- Incubation (1):

The plate was covered with the plate cover and incubated for 30 minute at 37℃.

Step 6- Washing (1):

After the end of incubation, the plate cover was removed and each well was washed 5 times with diluted wash buffer (Tween 20). Each time, allowing the microwells 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:

After that 100µl HRP-Conjugate was added to each well except the blank.

Step 8- HRP-Conjugate Incubation (2):

The plate was covered with the plate cover and incubated for 30 minute at 37℃.

Step 9- Washing (2):

At the end of incubation, the plate cover was removed and washed 5 times with diluted wash buffer as in step 6.

Step 10- Colouring:

Then 50µl of chromogen A and 50µl chromogen B solution was dispensed into well including the blank and mixedby tapping the plate gently,incubated for 15 minutes at 37℃ avoiding light.

Step 11- Stopping Reaction:

Using a multichannel pipette 50µl stop solution 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 540nm, and calculated the cut-off value and evaluated the results.

3.8.3 Calculation of cut off:

Cut off (C.O.) = mean NC+0.12

3.8.4 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

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

3.9 Statistical analysis

Data was analyzed using SPSS software package (version 16 for windows 7). Using Pearson chi -square test to determine the difference among various categories with respect to HCV seropositivity. A p value of <0.05 was considered statistically significant.

4. Results

This study was carried out during the period from February to May 2015 including 90 diabetic patients with age range from 45 years – 65 years.

From the 90 diabetic patients, 5 (5.5%) found to have anti-HCV antibodies within age group 45-55 years, Table (1) shows frequency of anti-HCV among age group above 45 years andgender with positive results, 3 were males and 2 were females 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**
Male	52	3	3.3%
Female	38	2	2.2%
Total	90	5	5.5%

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

Age groups	**No. of patients**	**No. of anti-HCV positive**	**Percentage of positive**

45-55 years	43	5	5.5%
56-65 years	47	0	0%
Total	90	5	5.5%

Table (3) shows correlation between the presence of HCV antibodies (Abs) with smoking, and only one patient was smoker and 4 patients were non-smoker.

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

Smoking	**HCV positive results**		**HCV negative results**		**Total**
	Frequency	**Percent**	**Frequency**	**Percent**	
Smoker	1	1.1%	9	10.0%	10
Nonsmoker	4	4.4%	76	84.4%	80
Total	5	5.5%	85	94.4%	90 (100%)

Chi-square test *P* value (0.453)

Table (4) shows the correlation between the presence of HCV Abs and family history of DM, it was found that 4 of the five positive patients were 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 results**		**Total**
	Frequency	**Percent**	**Frequency**	**Percent**	

History	4	4.4%	46	51.1%	50
No history	1	1.1%	39	43.3%	40
Total	5	5.5%	85	94.4%	90 (100%)

Chi-square test *P* value (0.258)

5. Discussion

5.1 Discussion

This study had determined the frequency of HCV among diabetic patients type2 attending Zinam hospital and to correlate between the presence of anti HCV Abs and other factors like gender, age, familyhistory of diabetes and smoking.

In this study the results showed that out of 90 diabetic samples 5 (5.5%) were positive for hepatitis C virus infection (HCV) within age group 45-55 years. Four of these positive patients showed family history of diabetes, two of these positive results were females and three were males one of them was smoker.

Different results were obtained in other studies, the present study results were higher compared with that carried out 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 2usingELISA fourth generation for anti- HCV antibodies was done in 180 samples of patients with Type 2 DM visiting El-RoseiresHospital, and 180 volunteer blood donors visiting blood bank of the same hospital, reported that 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, 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 without family history of diabetes showed a higher seroprevalence of 13 (6.7%) (Ndako*et al.*, 2009).

A cross sectional study carried out indifferent hospitals of Faisalabad amongst 154 Pakistanis diabetic patients show 18.83%(Yahya and Iqbal, 2011), and 5 (10%) out of 50 diabetic Yemeni patients were seropositive for HCV antibodies measured by chromatographic and enzyme-linked immunosorbent assay (ELSIA) (Habib *et al*., 2014).

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

It is necessary to determine the prevalence of HCV among diabetic patients to increase awareness among general population and health care workers to prevent morbidity and increased costs associated with this infection in diabetes due to failure of treatment. Since the prevalence of diabetes is on the rise and is complicated by co-infection with HCV, the determination of relationship becomes even more important in this scenario so that it can be effectively managed.

5.2 Conclusion

This study showed that the frequency of HCV among diabetic patients type2 was 5/90 (5.5%) within age group 45-55 years, 3/5 (3.3%) males and 2/5 (2.2%) females.

The correlation between the results and other factors showed insignificant relation between smoking and history of diabetes.

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

5.3 Recommendation

- Diabetic patients should be screened regularly for HCV to control and minimize development of complications.
- Awareness program of hepatitis infection and transmission should be done to people who are in high risk.
- Further studies should be done with large sample size to get reliable results and to find out the relation between diabetes and hepatitis viral infection.

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