Physical Education, Health and Social Sciences

https://journal-of-social-education.org E-ISSN: 2958-5996

P-ISSN: 2958-5988

Prevalence of Hepatic and Renal Dysfunction in Individuals with Diabetes Mellitus: A Public Health Concern

Matiullah[1], Javid Sattar[2], Nazia Azam[3], Mustafa Kamal[4], Muhammad Saqib Khalil[5], Imran Khan[6*]

[1,2,3,4] Health services academy Islamabad

[5, 6] Sarhad Institute of Allied Health Sciences, Sarhad University of Science and information Technology

1. matiullah.qadar@gmail.com, 2. javedsattar088@gmail.com 3. nax_axam@hotmail.com

4. kamal5380554@gmail.com 5. Saqib.siahs@suit.edu.pk

6. Corresponding Author Email: Imran.siahs@suit.edu.pk

DOI: https://doi.org/10.63163/jpehss.v4i1.1142

ABSTARACT

Diabetes mellitus is one of the major non-communicable diseases and the prevalence is rising globally. Diabetes is a leading cause of preventable blindness in adults aged between 20-75 years. Diabetes is increasingly recognized as the leading cause of chronic renal failure and hepatic disease. The aim of the study was to determine the function of liver and kidney of diabetic patients. A total of 40 blood samples from the diabetic patients were selected and then categorized into age wise ranging from 40 to 80 years and gender wise distribution. Using the commercially available kits according to the manufacturer's instructions. Blood urea, creatinine, ALT, bilirubin and ALP was estimated by the Microlab-300 Labtech reagent kit on the selected samples. Our result concluded that the renel function of diabetic patient's age ranging above 40 to 60 were slightly elevated than normal and the age ranging from 61 to 80 were more affected. The liver function of diabetic patients showed normal results age ranging from 40 to 50 years and the age ranging from 61 to 80 are slightly elevated. The renal function of diabetic patients were more elevated as compare to liver function. It is further concluded that in gender wise distribution females were more affected than males during RFTs analysis. Similarly in LFTs analysis males were more affected is compared to females.

Keywords: *Liver function tests, Renal function tests, Creatinine, Alanine transaminase, Alkaline Phosphatase, Bilirubin and Blood Urea.*

INTRODUCTION

Diabetes Mellitus a metabolic disorder arises if body is incapable to synthesize sufficient insulin for metabolism, it could be due to improper insulin secretion, improper insulin action, or both. Persistent exposure to excessive glucose could be a dominant reason for cardiomyopathies nephropathies, retinopathies, neuropathies and a variety of different sorts of tissue injury. Diabetes mellitus additionally leads to vascular disease, hypertension, dyslipidemia, and obesity (Arshad *et al.*, 2017).

Diabetes mellitus is one of the major non-communicable diseases and the prevalence is rising globally. Type 2 diabetes is the most common form, accounting for 90% of all cases. The prevalence of diabetes worldwide was estimated to be 2.8% in 2000 and 4.4% in 2030. The total

number of diabetes is projected to increase from 171 million in 2000 to 366 million in 2030. Diabetes is more prevalent in men than women (Ni, *et al*., 2012).

Diabetes is a leading cause of preventable blindness in adults aged between 20 - 75 years. Among the ocular complications of diabetes, diabetic retinopathy is the most frequent and it is responsible for 12% of all new cases of blindness every year. Among patients of type-I and type-II diabetes for more than 20 years, the prevalence of diabetic retinopathy has been reported to be 95% and 60%, respectively. However, the prevalence varies depending upon the population and the age group being studied. Existing research has identified a number of factors attributable for the development of diabetic retinopathy including but not limited to duration of diabetes along with glycemic control, systemic hypertension, hyperlipidemia, obesity and positive family history of diabetes. Prevention and treatment of these attributing factors is as much important as good glycemic control to avoid diabetic retinopathy (Anwar, S. B *et al.,* 2019).

The number of diabetic patients is increasing at an alarming rate in developing countries that is approaching epidemic proportions. In diabetes mellitus, blood sugar levels are raised, increasing the risk of other complications like retinopathy, nephropathy, and neuropathy. Two types of diabetes mellitus are type 1 and type 2. In type 1 diabetes, insulin is not produced due to autoimmune destruction of b cells. In 2013, diabetes affected 382 million people, and numbers of those who live with this chronic disease are increasing continuously and, at this rate, are expected to reach 592 million in the next 22 years. Type 2 diabetes mellitus (T2DM) is a disorder of glucose metabolism in which the body becomes resistant to the glucose-lowering effects of insulin, resulting in hyperglycemia. Complications associated with diabetes are often attributed to hyperglycemia-induced oxidative stress and inflammation. Pakistan ranks among the top 10 countries with the highest burden of diabetes. In Pakistan, 6.4 million people were affected with diabetes in 2011, which is supposed to increase to about 11.4 million by 2030 (4) (Aslam, *et al.* 2019)

We know that diabetes affects the kidney in stages. At the very onset of diabetes, the kidney grows large and the glomerular filtration rate (GFR) becomes supranormal. Most recent basic and clinical research have slanted toward sclerosis and kidney failure that occur many years later. However, the original idea that the early hemodynamic phenotype provokes the subsequent demise of a diabetic kidney has not been abandoned. The Starling forces and capillary surface that determine GFR are directly influenced by various effectors that include nerves, hormones, size and condition of the glomerulus, and tubuloglomerular feedback (TGF) from the macula densa. Physiological effectors of GFR generally function as elements in negative feedback systems that link GFR to some other physiological parameter. For example, glomerular filtration is linked to extracellular fluid volume (ECF) through multiple parallel feedback loops that respectively incorporate the renal nerves, renin-angiotensin system (RAS), and natriuretic peptides (ANP). Glomerular filtration is linked to the concentration of salt in the tubular fluid that reaches the macula densa (MD [NaCl]) by TGF. GFR and size of the kidney are also linked in a negative feedback arrangement, although how a feedback signal derives from GFR that influences kidney growth remains a mystery. Effectors that influence tubuloglomerular feedback (TGF) of negative feedback loops that link glomerular filtration rate (GFR) to other physiological parameters. ECF, extracellular fluid volume; MD[NaCl], salt concentration of tubular fluid at the macula densa; Nerves, efferent sympathetic renal nerves; RAS, renin-angiotensin system; ANP, atrial natriuretic peptide hormones from the heart; solid lines, proportional effects; dashed lines, inverse effects. A negative feedback loop must contain an odd number of inverse effects (Thomson *et al*., 2004).

People with diabetes have nearly 2-fold higher odds of CKD than those without diabetes. The odds ratios for CKD vary between 1.3 and 4.6, depending on the region of the world, and this risk is compounded by the presence of hypertension. Among those with diabetes, CKD prevalence varies

widely between countries with estimates ranging from 27.1% in Shanghai, China to 83.6% in Tanzania. In the US National Health and Nutrition Examination Survey (NHANES) 2009 to 2014, CKD prevalence was 26.2% among adults with diabetes, taken into account albuminuria persistence. In those aged 65 years low eGFR was significantly more prevalent than among younger age groups, whereas persistent albuminuria prevalence was comparable. CKD in patients with diabetes, like in other diseases, is clinically defined by the presence of persistent albuminuria (albumin-tocreatinine ratio [ACR] 30 mg/g for at least 3 months) and/or persistent low estimated glomerular filtration rate (eGFR ,60 mL/min/1.73 m2) regardless of etiology. Certain risk factors for diabetic kidney disease are important targets in the prevention or delay of CKD and for personalizing treatment strategies. Genetic factors, male sex, age, and duration of diabetes, are among the no modifiable risk factors associated both with onset and progression of kidney disease. The modifiable risk factors include poor glycemic control, hypertension, lipid abnormalities, smoking, obesity, insulin resistance, low intensity of physical activity, high salt intake, birth weight, exposure to diabetes in utero, and periodontal disease (Koye, *et al.,* 2018).

Diabetes is increasingly recognized as the leading cause of chronic renal failure, with many patients progressing to end stage renal disease (ESRD) requiring dialysis or transplantation. Diabetic kidney disease (DKD) also referred as diabetic nephropathy (DN) is clinically characterized by a progressive increase in albuminuria and a subsequent decline in glomerular filtration rate (GFR). This disorder is often accompanied by a disproportionate decrease in afferent arteriole resistance while there is an increase in efferent arteriole resistance, with the resulting intra-glomerular hypertension causing further damage to the kidney, and ultimately leading to end stage renal failure. The pathogenesis and clinical manifestations of DKD usually follow a predetermined course that is associated with marked structural changes in the kidney, including renal hypertrophy, enlargement of glomerular capillaries, mesangial expansion and glomerular basement membrane (GBM) thickening due to an excessive deposition of extracellular matrix (ECM). The development and progression of DKD is a complex process due to the wide diversity of cell populations present within the kidney and the various physiological roles of this organ. Chronic hyperglycemia leads to the activation of several pathological processes which affect numerous resident renal cells, including the glomerular endothelial cells, smooth muscle cells, mesangial cells, podocytes, and cells of the tubular and collecting duct. Accumulating evidence has now implicated these key renal cell-types in driving the structural and functional changes in diabetic kidneys. Possessing a crucial role in maintaining normal glomerular capillary permeability, endothelial cells and podocytes become structurally immature with diminution of the endothelial glycocalyx in hyperglycemic conditions. Of importance, ultra-glomerular structural changes including changes in podocyte structure and function are closely related to an increase in albuminuria in DKD (Jha, *et al*., 2016).

The kidney frequently gets involved in type 2 diabetes, starting with signs of hyperfiltration followed by microalbuminuria (>30 mg of albumin in the urine per 24 hr). The presence of microalbuminuria occurs in 30% to 50% of patients with type 1 diabetes, and it is estimated to occur in approximately 20% to 30% of all patients with type 2 diabetes at the time of diagnosis. The progression to further involvement of the kidney is shown when urinary albumin excretion rises to macroalbuminuria (> 300 mg/24 hr), which occurs in ~3 % of the microalbuminuric type 2 diabetic patients per year. Patients with macroalbuminuria are at risk of progressive renal function decline and loss of hormonal functions that are regulated by the kidney. These functions include volume homeostasis, blood pressure, vitamin D metabolism, and erythropoietin production. Finally, ~10% of patients who survived death (mostly) from cardiovascular disease end up in dialysis or with transplantation (Heerspink, *et al*., 2016).

The liver has a major role in glucose homeostasis and, in liver diseases, hepatic carbohydrate

metabolism is commonly disturbed. Altered portal insulin levels and the insulin/glucagon ratio may influence hepatocyte function and integrity in diabetic patients and predispose them to various hepatic disorders. Although no specific liver disease is known to be associated with diabetes mellitus, altered hepatic glucose metabolism may be involved in the pathogenesis of non-insulin-dependent diabetes. Disturbances in liver function tests (LFT) are well recognized in some diabetic patients, especially in acute metabolic decompensation. However, in diabetic patients, the prevalence of abnormal LFT results and their relationships to clinical findings and diabetes per se, as well as to pathologic changes in liver structure, are controversial (Salmela, *et al*., 1984).

Hepatocellular glycogen accumulation leads to hepatomegaly and liver enzyme abnormalities in poorly controlled diabetes patients. In hyperglycemic states, there will be intracellular glycogen accumulation in the hepatocytes due to increased glycogen synthesis, causing typical biochemical findings of mild to moderately elevated aminotransferases, normal liver synthetic function, with or without mild elevations of alkaline phosphatase. All these biochemical disturbances and hepatomegaly are found to be reversible with good glycemic control. Serum aminotransferases such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST) indicate the concentration of hepatic intracellular enzymes that have leaked into the circulation. These are the markers for hepatocellular injury and are used as primary screening of non-alcoholic steatohepatitis (NASH). Chronic mild elevations of ALT and AST are seen in type 2 diabetes patients. In a study done in United States by Erbey et al in 2000, the prevalence of elevated ALT among type 2 diabetes is 7.8% compared to 3.8% in those without diabetes. In another study by Salmela *et al*, elevated ALT in diabetes patients was associated with increased BMI and poor glycemic control in multivariate analysis (Ni, *et al*., 2012).

Liver function tests (LFTs) are commonly used in clinical practice to screen for liver disease, monitor the progression of known disease, and monitor the effects of potentially hepatotoxic drugs. The most common LFTs include the serum aminotransferases, alkaline phosphatase, bilirubin, albumin, and prothrombin time. Aminotransferases, such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST), measure the concentration of intracellular hepatic enzymes that have leaked into the circulation and serve as a marker of hepatocyte injury. Alkaline phosphatase (AP), γglutamyl transpeptidase (GGT), and bilirubin act as markers of biliary function and cholestasis. Albumin and prothrombin reflect liver synthetic function. The aminotransferases AST and ALT are normally < 30 -40 units/l. Elevations of aminotransferases greater than eight times the upper limit of normal reflect either acute viral hepatitis, ischemic hepatitis, or drug- or toxin-induced liver injury.

Chronic mild elevation of transaminases is frequently found in type 2 diabetic patients. This article will provide a review of the pathology, incidence, causes, and drug therapy related to type 2 diabetic patients with elevated LFTs. GGT is a nonspecific marker that is known to rise in patients with type 2 diabetes. In epidemiological studies, it has a positive association with alcohol intake, cigarette smoking, coronary heart disease, BMI, systolic blood pressure, serum triglyceride, heart rate, uric acid, and hematocrit. It has an inverse association with physical activity level. Because GGT increases in diabetes, and increases as BMI increases, it has been proposed as another marker of insulin resistance. LFTs measured included albumin, total bilirubin, AST, ALT, AP, GGT, and serum concentrations of colic acid and chenodeoxycholic acid. Fifty-seven percent of the 175 diabetic outpatients (100 subjects) had at least one abnormal LFT; 27% (48 subjects) had at least two abnormal tests. The type 2 diabetic patients more frequently had elevated ALT (22.9 vs. 5.3%) and GGT (23.7 vs. 10.5%) levels than those with type 1 diabetes. On the other hand, patients with type 1 diabetes more frequently had elevated bilirubin levels (21.1 vs. 10.2%). However, increases in LFTs were rarely more than twice the upper limit of normal. The most common cause of elevated LFTs in type 2 diabetic patients is nonalcoholic fatty liver disease (NAFLD). NAFLD is a

clinicopathological condition representing a spectrum of histological findings from hepatic steatosis or fat accumulation in hepatocytes without inflammation, to hepatic steatosis with a necroinflammatory component that may or may not have fibrosis, or NASH (Harris *et al.,* 2005).

METHODOLOGY

Study place:

This study was conducted in public Hospital ―Institute of Kidney Disease Hayatabad Peshawar‖ in district Peshawar KP.

Study Duration:

The study lasted for 45 Days.

Sample size:

A total of 40 subjects (diabetic patients) were selected for study, ages ranging from above 40 years.

Selection criteria:

Sampling was done according to the following criteria.

Inclusion criteria

All diabetic type 1 and type 2 males and females patients are included in this study.
Diabetic patients above 40 years are included.

Exclusion criteria

Diabetic patients less than 40 years are excluded from this study. Diabetic Patients with CVD are also excluded.

Sample collection processing

After the approval of research proposal by the concerned members of the Undergraduate Research committee of Sarhad Institute of Allied Health Sciences. An informed consent was taken from the administration of Hospital. After taken of the consent form the subject about 3 ml of venous blood was withdrawn from each subject by using a disposable plastic syringe after sterilization of skin with isopropyl alcohol (70%) swabs. This sample was withdrawn to gel tube. Blood left to be clotted in a gel tube for 30 min in the incubator then centrifuged for 15 min at approximately 3000 rpm. The expressed serum was used for determination of serum Creatinine urea level and ALT, serum Bilirubin and ALP on Microlab-300 (Germany) was used for the investigation of blood metabolic products respectively. The results was confirmed by automation examination after confirmation the result (data) about the diabetic patients from whom samples were collected recorded by filling a Performa and at the end of examination, the original results data were kept saved by us and copies of them were provided to the head of the Hospital laboratory. The positive cases were advised to show them to the doctor for treatment.

Estimation of waste metabolites:

Using auto-analyzer, Micro-lab-300 clinical chemistry automation systems. Using the commercially available kits according to the manufacturer's instructions. Blood urea, creatinine, ALT, bilirubin and ALP was estimated by the Micro-lab-300 Labtech reagent kit.

Principal:

The basic principal of Micro-lab-300 clinical chemistry automation is Beer Lambert Law.

Beer Lambert Law:

Beer Lambert Law helps us define the relationship between the intensity of visible UV radiation and the exact quantity of substance present. The derivation of Beer Lambert Law has many applications in modern day science. Used in modern day labs for testing of medicines, organic chemistry and tests with quantification. These are some fields in which this law finds its uses.

The Beer-Lambert law states that **—for a given material sample path length and concentration of the sample are directly proportional to the absorbance of the light‖**.

The Beer-Lambert law is expressed as: $A = \varepsilon Lc$

Where,

- A is the amount of light absorbed for a particular wavelength by the sample
- ε is the molar extinction coefficient
- L is the distance covered by the light through the solution c is the concentration of the absorbing species.

Test procedure:

Creatinine:

- Wavelength…….492nm. (490 - 510) Cuvette……. 1cm light path.
- Temperature……37°C. 15 - 25°C
- Adjust the instrument to zero with distilled water.
- Pipette into a cuvette:
- Prepare the necessary materials and reagents:
  - R1 (Reagent 1) - 500 µL
  - R2 (Reagent 2) - 500 µL
  - Serum - 100 µL
- Mix and start stop watch.
- Read the absorbance A_1 after 30 seconds and after 90 seconds A_2 of the sample addition.
- **Serum Creatinine** Normal Range: 0.6 - 1.1 mg/dl.

Urea:

- Wavelength…….340nm.
- Cuvette……. 1cm light path.
- Temperature……37°C. 15 - 25°C
- Adjust the instrument to zero with distilled water.
- Pipette into a cuvette:
- Prepare the necessary materials and reagents:
  - R1 (Reagent 1) - 800 µL
  - R2 (Reagent 2) - 200 µL
  - Serum - 20 µL
- Mix and Read the absorbance A_1 after 30 seconds and after 90 seconds A2.
- **Serum Urea** Normal Range: 11 - 40 mg/dl.

Alkaline Phosphatase (ALP):

- Wavelength…….405nm.
- Cuvette……. 1cm light path.
- Temperature……25°C/30°C/37°C
- Adjust the instrument to zero with distilled water.

- Pipette into a cuvette:
- Prepare the necessary materials and reagents:
  - R1 (Reagent 1) - 1000 µL
  - R2 (Reagent 2) - 200 µL
  - Serum - 10 µL
- Mix and incubate for 1 minute.
- Read the absorbance $A_{1\ of}$ the sample start the stop watch and read absorbance at 1 min. interval thereafter for 3 min.
- **Alkaline Phosphatase** ALP Normal Range: less than < 258 U/l **Total Bilirubin:**
- Wavelength…….555 nm (530 - 580) □ Cuvette……. 1cm light path.
- Temperature……..…15 - 25°C
- Adjust the instrument to zero with distilled water.
- Pipette into a cuvette:
- Prepare the necessary materials and reagents:
  - R1 (Reagent 1) - 1500 µL
  - R2 (Reagent 2) - 50 µL
  - Serum - 100 µL
  - Mix and incubate exactly for 5 minutes at 15-25°C.
  - Read the absorbance (A).
  - **Serum Bilirubin** Normal Rang: less than < 1.1 mg/dl **Alanine Transaminase (ALT)**
  - Wavelength.....340nm.
  - • Cuvette..... 1cm light path.
  - • Temperature.....37°C.
  - • Adjust the instrument to zero with distilled water.
  - • Pipette into a cuvette:
  - • Prepare the necessary materials and reagents:
    - R1 (Reagent 1) - 800 µL
    - R2 (Reagent 2) - 200 µL
    - Serum - 100 µL
- • Mix and start stop watch.
- • Read the absorbance (A) after 60 seconds.
- **ALT** normal range up to 40 U/l

RESULTS

Age wise distribution of Patients

In this study total of n=40 Diabetic patients of age above 40 years male and female were examined. All the patients divided into four groups according to its age. The first group A whose age between 40 to 50 years consist of n=13 (32.5%) diabetic patients. The second group B whose age from 51 to 60 years consist of n=12 (30%) diabetic patients. The third group C whose age from 61 to 70 years consist of n=12 (30%) diabetic patients. The forth group whose age from 71 to 80 years consist of n=3 (7.5%) diabetic patients. Shown in Table.1

Table 1 Age wise distribution of individuals

Age Groups	No of Samples	Percent	Cumulative Percent
40 to 50 Years	13	32%	32%
51 to 60 Years	12	30%	62%
61 to 70 years	12	30%	92%
71 to 80 Years	3	8%	100%
Total	40	100%	

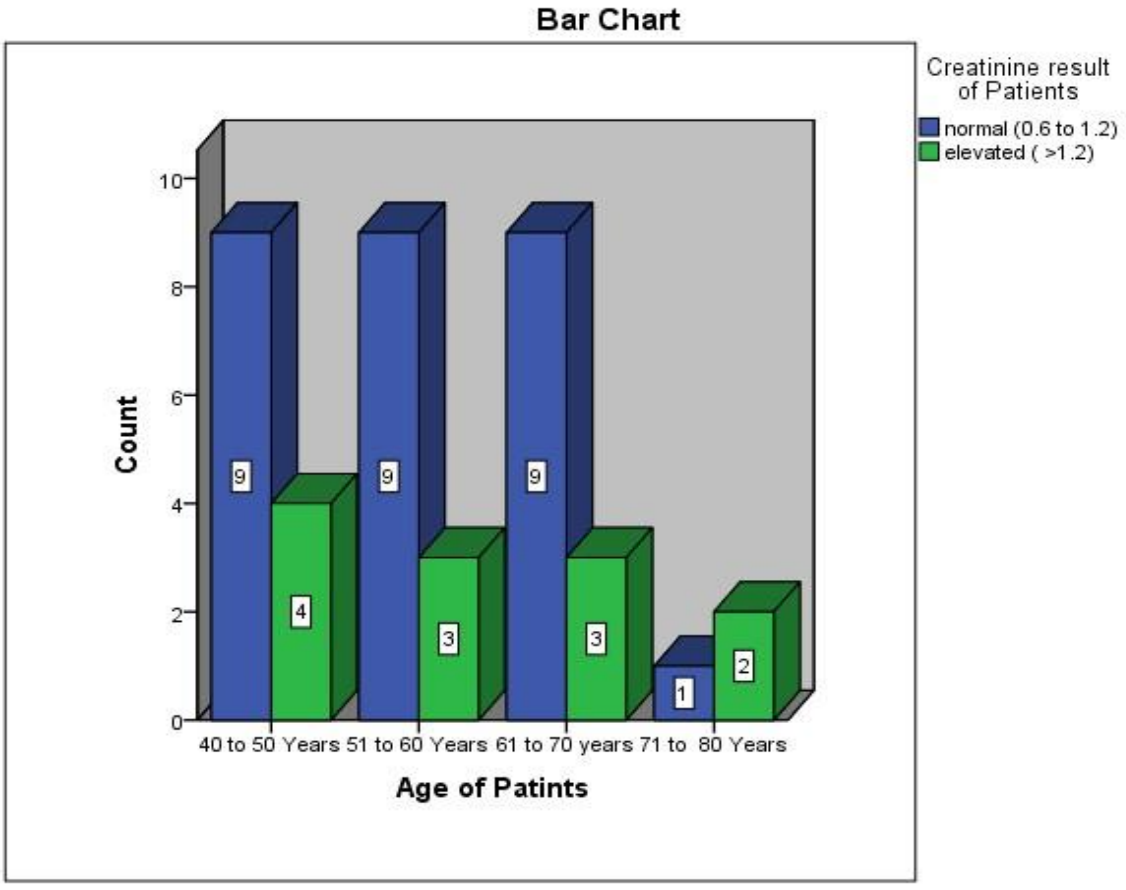

Fig. 1: Level of creatinine in blood sample of diabetic patients

Fig.1 showed that total 40 samples of diabetic patients were collected which were ranged at the age of 40 to 50, 51 to 60, 61 to 70 and 71 to 80. After analysis it was observed that the range at the age of 40 to 50 year total 13 samples were collected 9 (70%) were normal and 4 (30%) were elevated. Similarly at the age of 51 to 60 total 12 samples were collected 9 (75%) were normal and 3 (25%) were elevated. Likewise the next age group which ranges from 61 to 70 total 12 sample were taken in which 3 (25%) were elevated and 9 (75%) were normal. And the age group ranges from 71 to 80 total sample size was 3 in which 2 (67%) were infected and 1 (33%) indicates as normal.

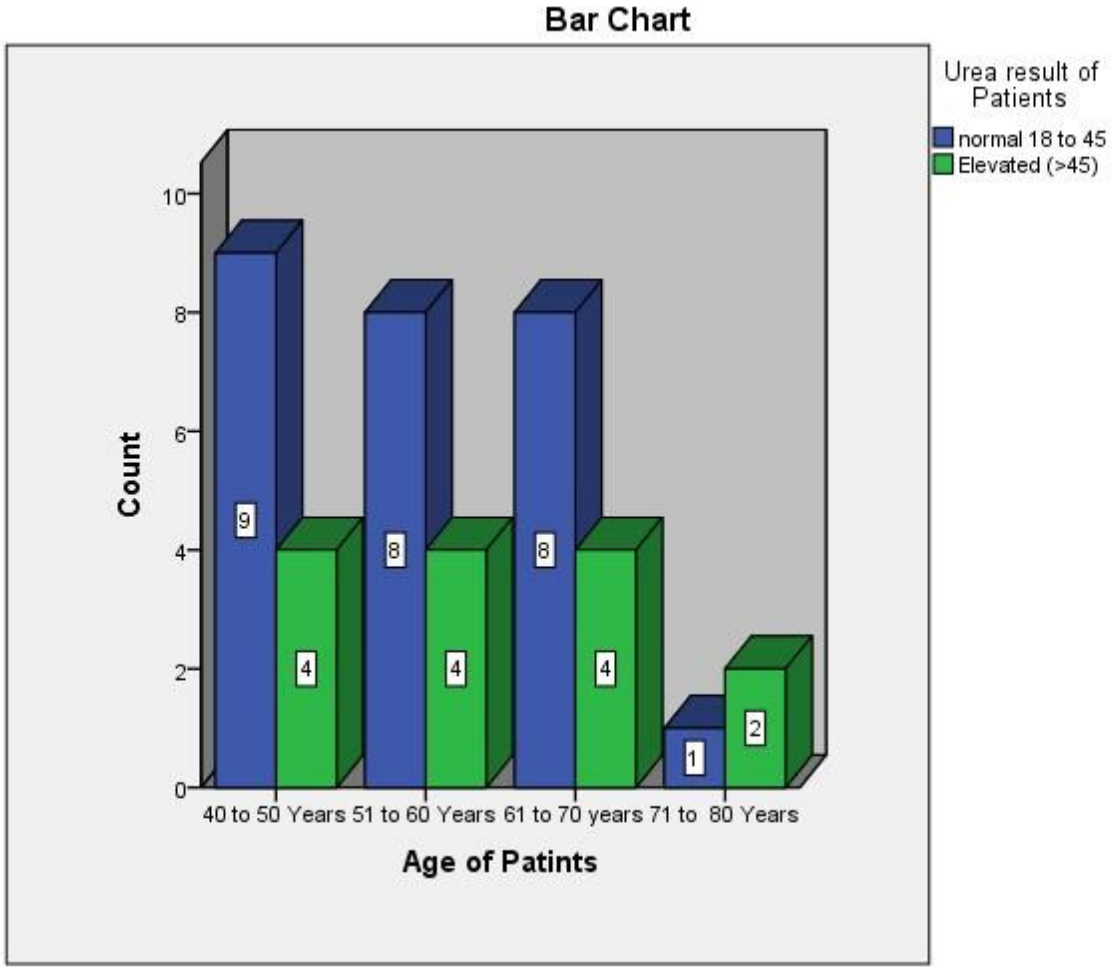

Fig. 2**:** Level of Urea in blood sample of diabetic patients

Figure 2 shows the collection of 40 samples from diabetes patients ranging in age from 40 to 50, 51 to 60, 61 to 70, and 71 to 80. Analysis revealed that the age range between 40 and 50 total 8 (61%) of the 13 samples were normal, and 4 (39%) had elevated levels. Similarly, 12 total samples were taken between the ages of 51 and 60, of which 8 (66%) were normal and 4 (33%) were abnormal. Likewise, 12 samples from the age group of 61 to 70 were obtained in total, of which 4 (33%) had increased levels of urea and 8 (66%) had normal levels. The age range is 71 to 80, and the sample size was three, of whom 2 (66%) were elevated and 1 (33%) was normal.

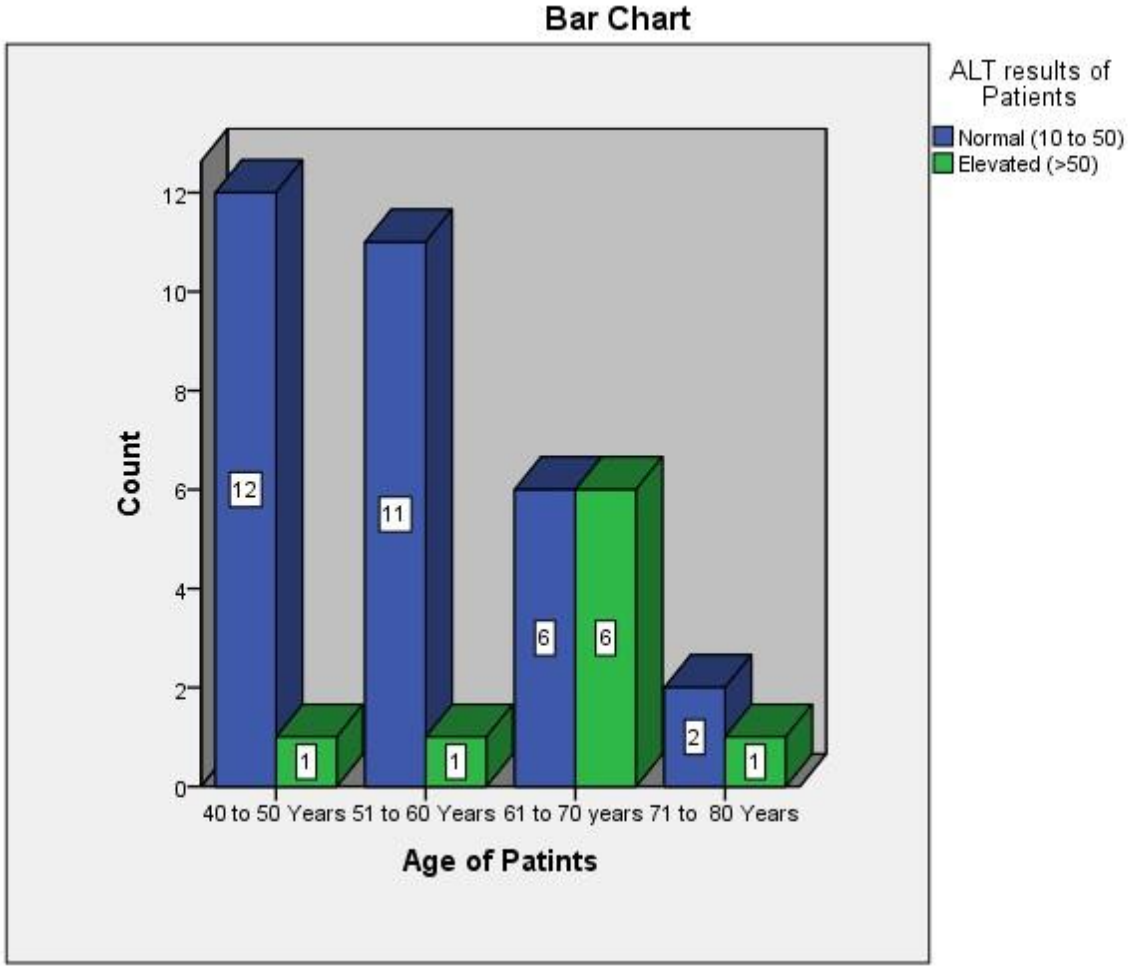

Fig. 3: Level of ALT in blood sample of diabetic patients

Figure 3 indicates the collection of 40 samples from patients with diabetes who were between the ages of 40 and 50, 51 to 60, 61 to 70, and 71 to 80. 12 (92%) of the 13 samples, representing the age range between 40 and 50, were found to be normal, and 1 (8%) had elevated levels after analysis. Similarly, a total of 12 samples between the ages of 51 and 60 were collected, of which 11 (91%) were considered to be normal and 1 (9%) to be abnormal. Likewise, a total of 12 samples from the 61 – 70 age range were collected, of which 6 (50%) had elevated levels and 6 (50%) had normal levels. The 3 people in the sample, 1 (33%) of whom had elevated levels of ALT and 2 (66%) of whom had normal levels, ranged in age from 71 to 80.

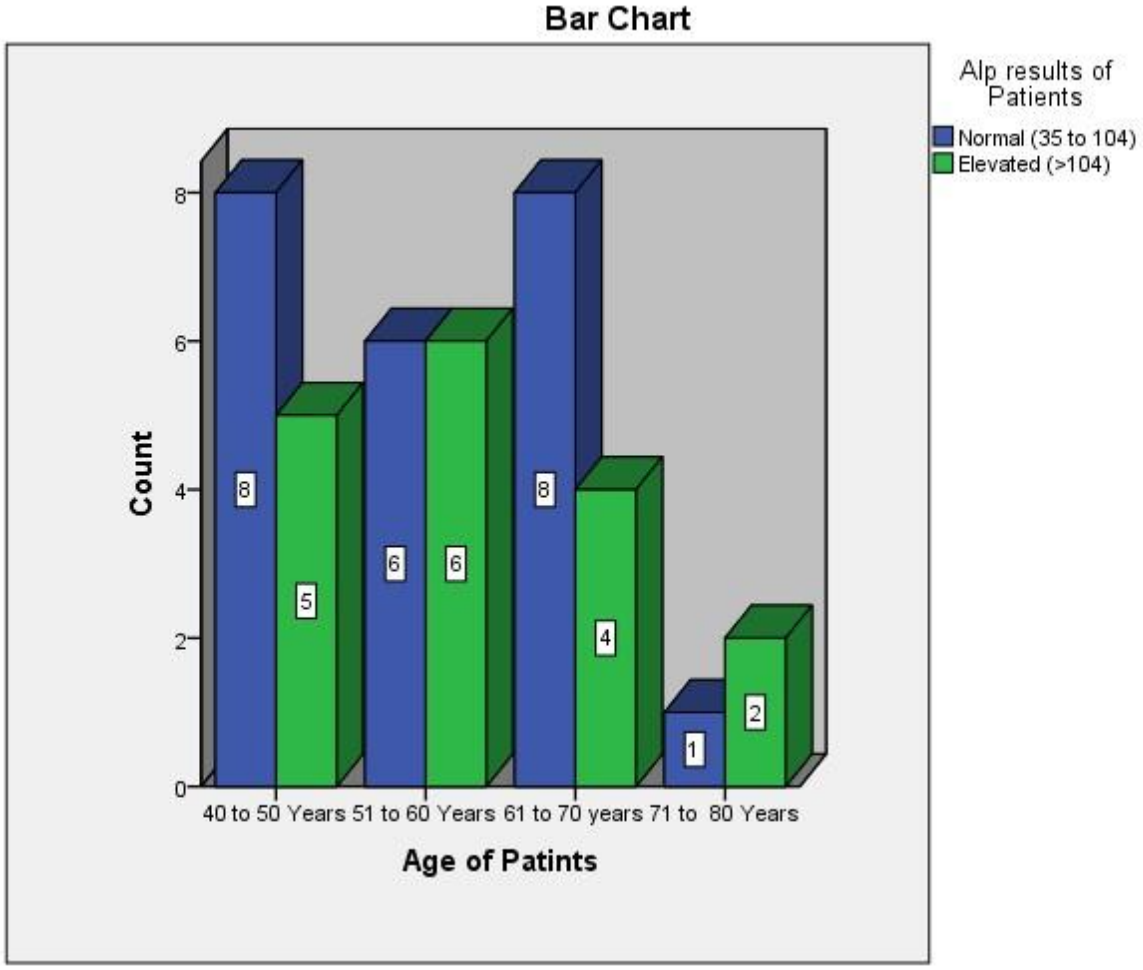

Fig. 4: Level of ALP in blood sample of diabetic patients

The collection of 40 samples from diabetic patients aged 40 to 50, 51 to 60, 61 to 70, and 71 to 80 is shown in Figure 4.4. After investigation, 8 (61%) of the 13 samples representing the 40 – 50 age range—were revealed to be normal, and 5 (39%) showed abnormal levels. Similar to this, 12 samples overall between the ages of 51 and 60 were extracted; 6 (50%) of them were considered to be normal, while 6 (50%) were considered to be high. Similar to this, 12 samples overall from people aged 61 to 70 were gathered, of which 8 (66%) had increased levels and 4 (33%) had normal levels. 2 (66%) of the 3 sample participants had elevated levels, whereas the other 1 (33%) had normal levels. They ranged in age from 71 to 80.

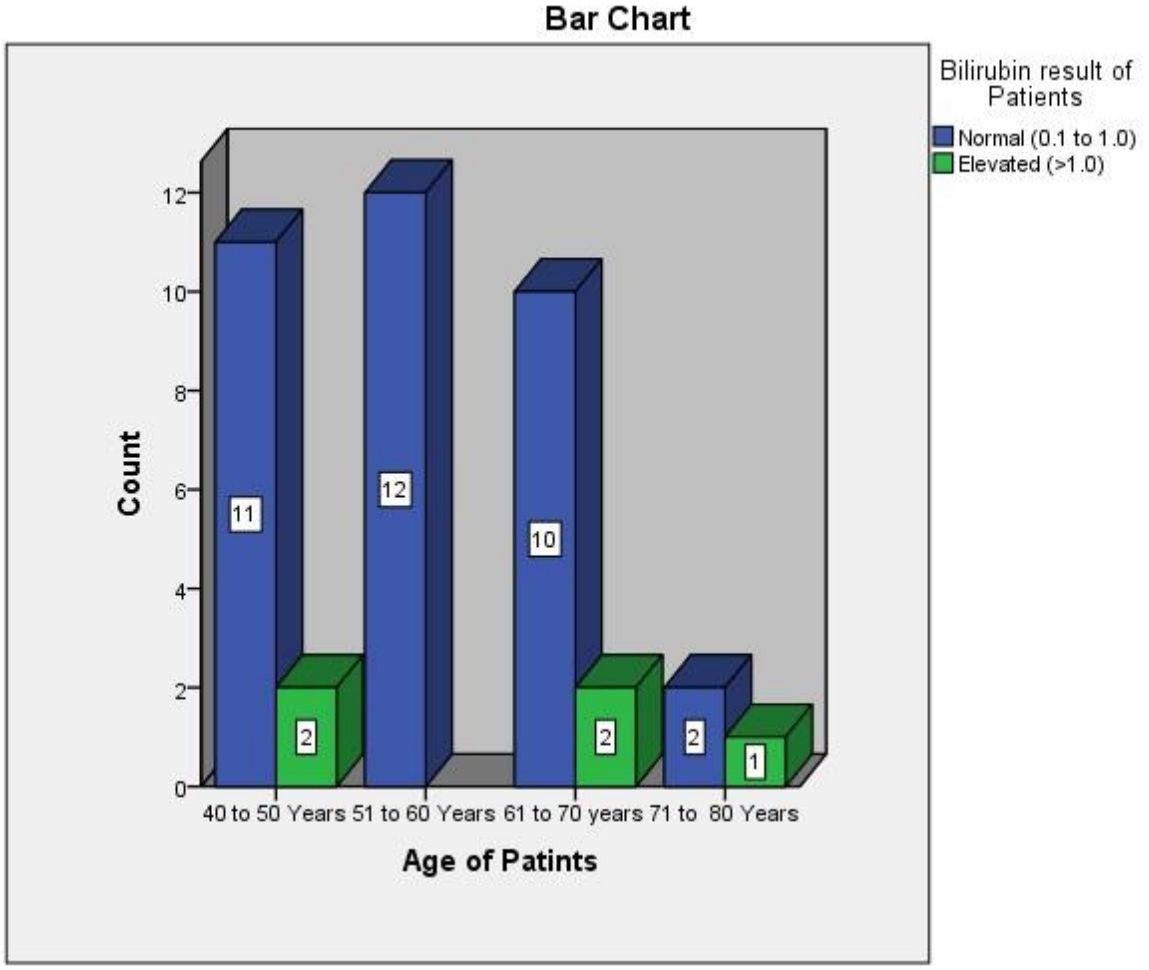

Fig. 5: Level of Bilirubin in blood sample of diabetic patients

Figure 5 represents the collection of 40 samples from diabetes patients between the ages of 40 to 50, 51 to 60, 61 to 70, and 71 to 80. 11 (84%) of the 13 samples representing the 40 – 50 age range were found to be normal after review, and 2 (16%) showed elevated values. Similar to this, a total of 12 samples between the ages of 51 to 60 were collected; all of them were determined to have normal levels of serum bilirubin. Similar to this, a total of 12 samples from patients between the ages of 61 to 70 were collected, of which 10 (83%) contained elevated levels and 2 (17%) had normal levels. The levels of 1 (33%) of the 3 sample participants were increased level of bilirubin, while the levels of the 2 (66%) participants were normal. They were between 71 and 80.

Gender wise distribution of Patients

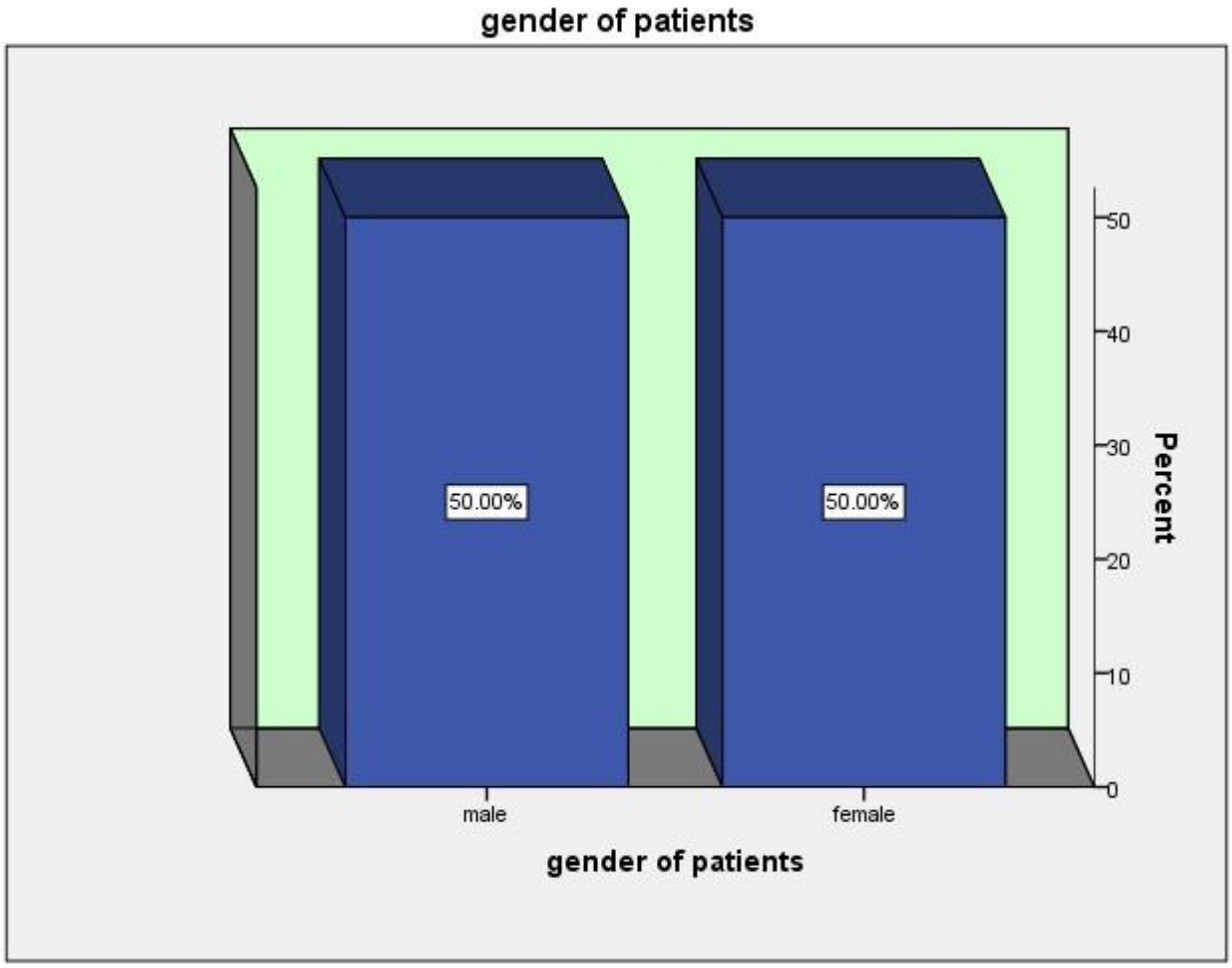

Fig. 6: Gender wise distribution of diabetic patients

In this study, a total of 40 diabetic patients over 40 years old, including both male and female participants, were examined. This contained 20 (50%) male and 20 (50%) female.

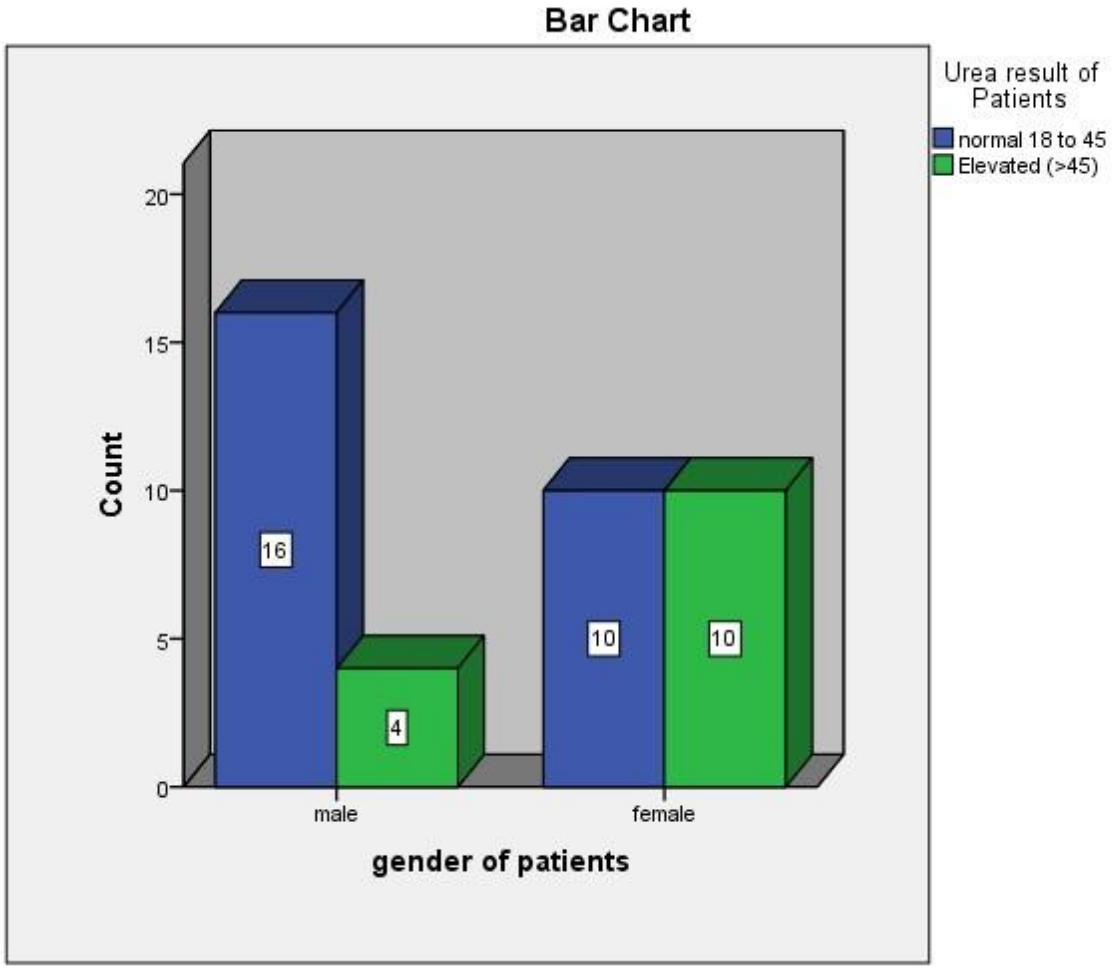

Fig. 7: Gender wise evaluation of urea levels in diabetic patients

The fig.7 showed a total of 40 diabetes patients, over the age of 40, in which 20 (50%) of them were male and 20 (50%) were female from the investigation it came to know from the 20 samples of males, 16 (80%) were found to be normal and 4 (20%) of them were above normal. Similarly, out of 20 samples 10 (50%) of females the level of urea were high from the normal level and the rest of 10 (50%) were normal

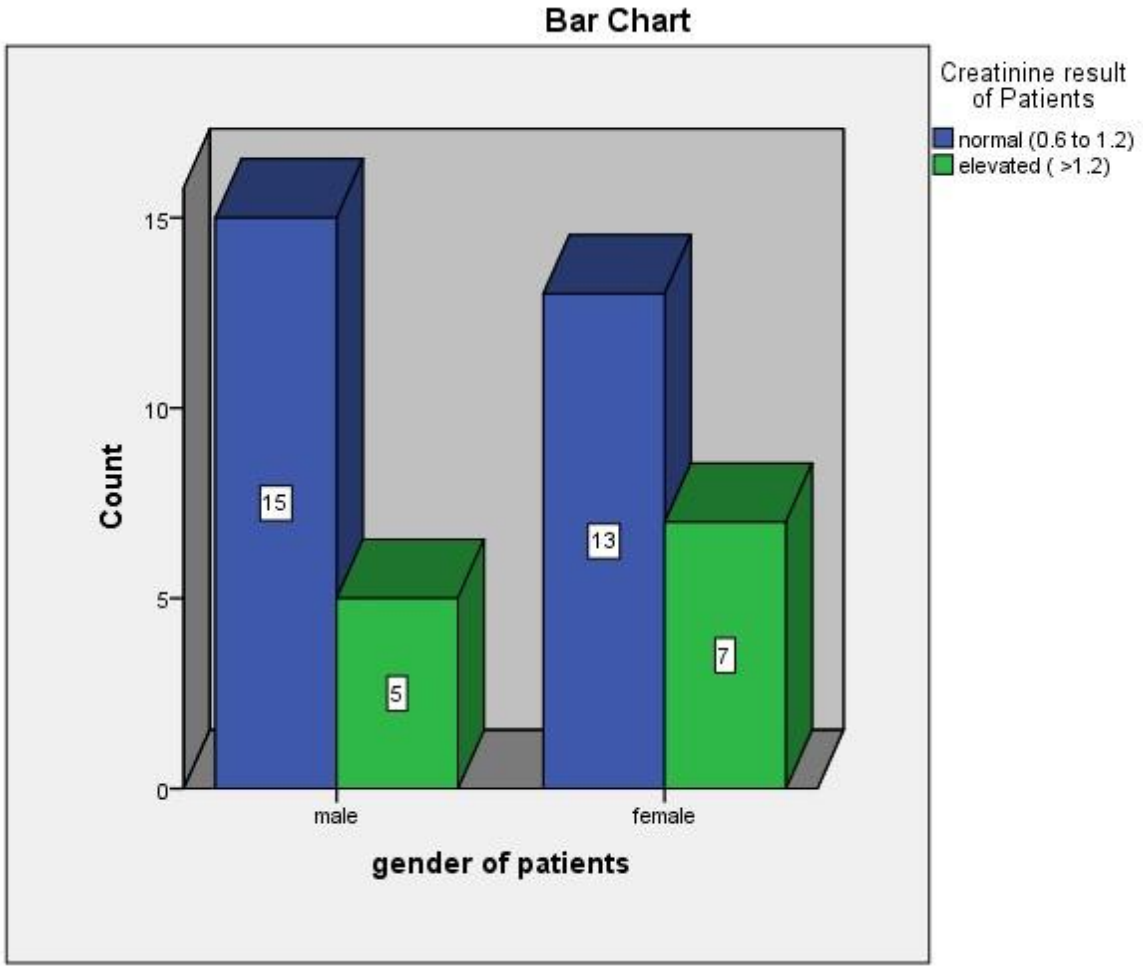

Fig. 8: Gender wise evaluation of creatinine in diabetic patients

Figure 8 shows that the evaluation of 40 diabetes patients over the age of 40, in which 20 (50%) male and 20 (50%) female patients, was researched. Following an assessment, it was discovered that 15 (75%) of the 20 samples from males were normal and 5 (25%) were higher than normal. Similar to this, 13 (65%) of the 20 female samples were normal, whereas 7 (35%) of them had elevated levels.

Fig. 9: Gender wise evaluation of ALT levels in diabetic patients

Figure 9 demonstrates that a study was conducted on 40 diabetes patients over the age of 40, including 20 (50%) male and 20 (50%) female patients. Following the analysis, it was found that 6 (30%) of the 20 male samples were elevated and 14 (70%) of the 20 samples were normal. Similar to this, 3 (15%) of the 20 female samples exhibited increased ALT levels, whereas 17 (75%) of them were normal.

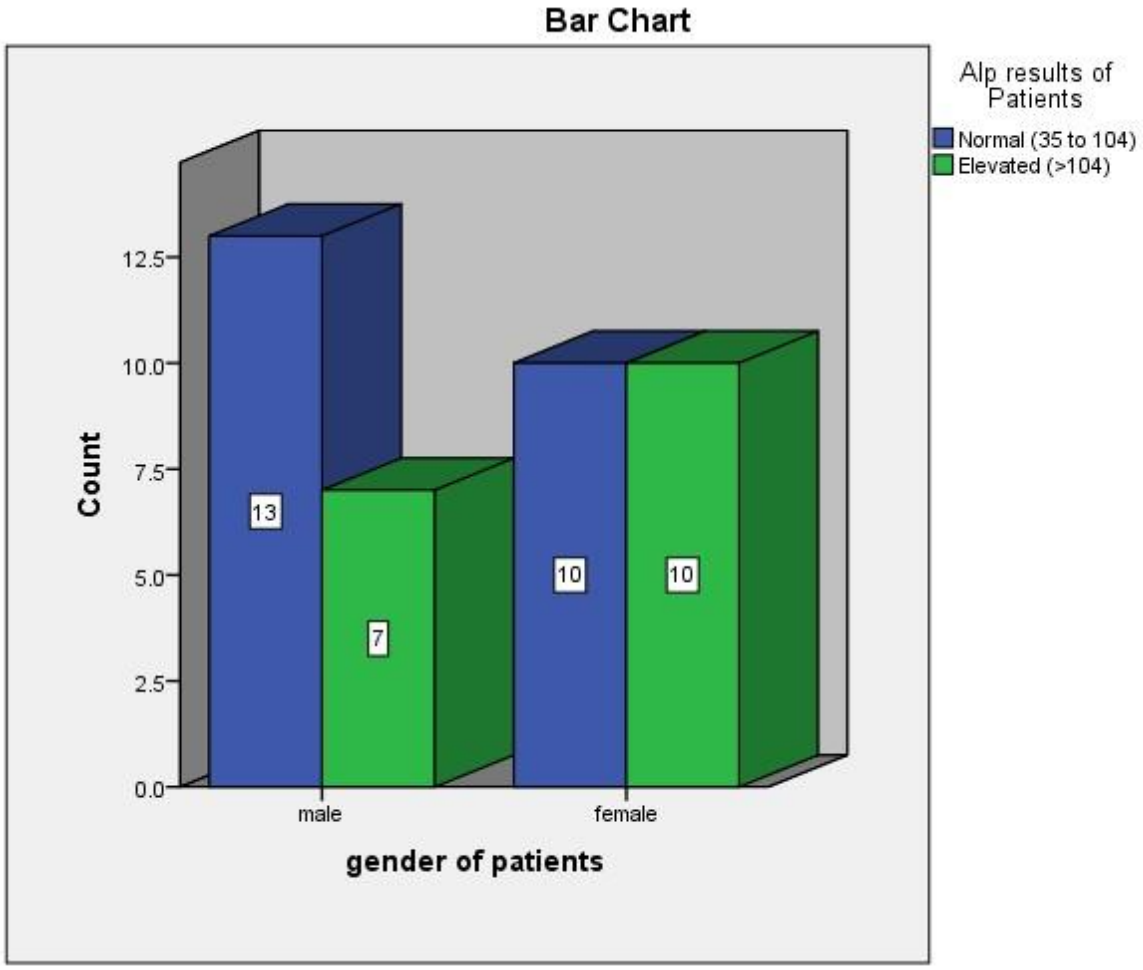

Fig.10: Gender wise evaluation of ALP levels in diabetic patients

Figure 10 shows that a study of total 40 diabetic patients involving 20 (50%) male and 20 (50%) female patients who were over the age of 40 was carried out. Following investigation, it was analyzed that 13 (65%) out of the 20 male samples were normal, while 7 (35%) out of the 20 male samples had abnormal values. Similar to this, 10 (50%) of the 20 female samples showed elevated levels, while only 10 (50%) showed normal levels.

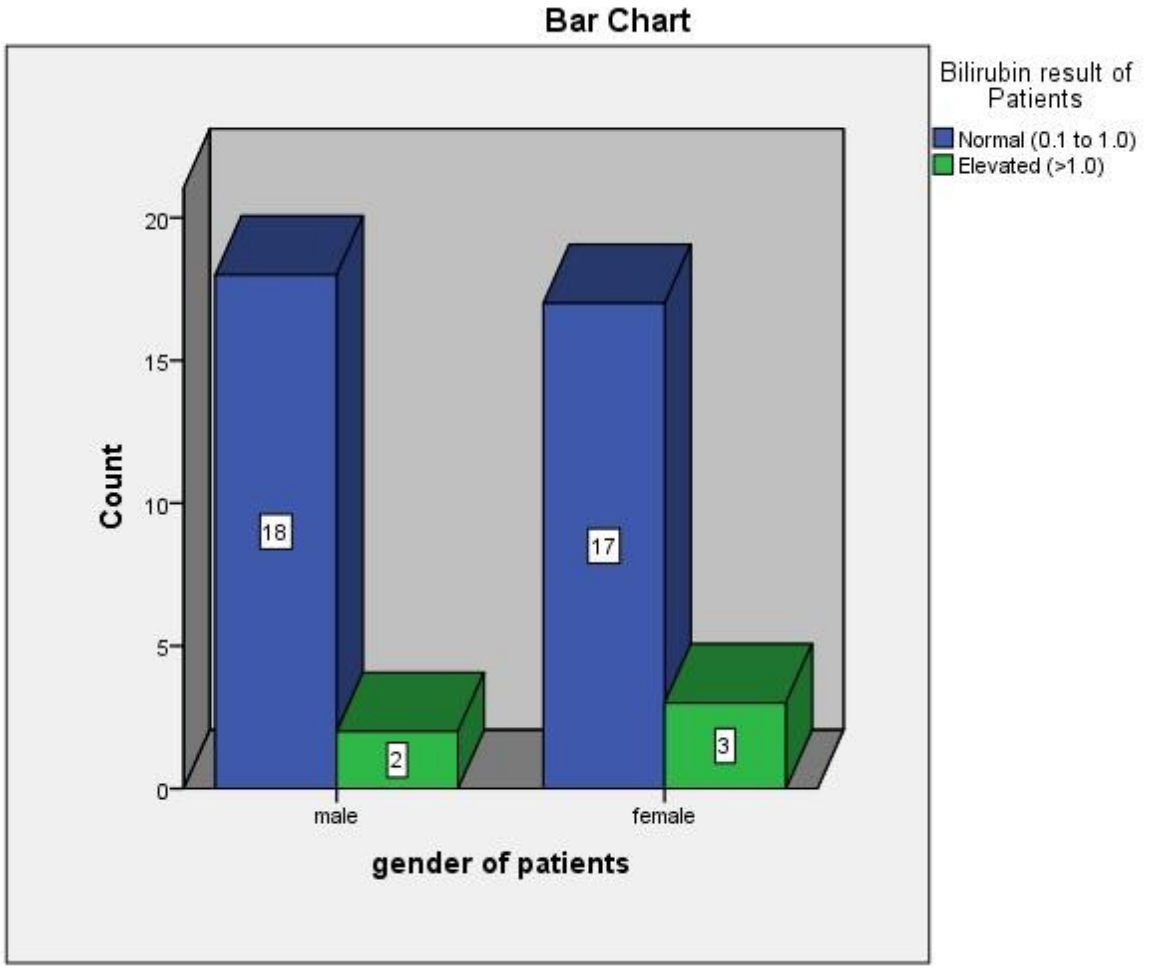

Fig.11 Gender wise evaluation of bilirubin in diabetic patients

Fig 11 Represents the outcomes of a study in which a total of 40 diabetes patients were chosen, 20 (50%) of whom were male and 20 (50%) of whom were female older than 40. Investigation showed that 2 (10%) out of the 20 male samples had abnormal values, while the remaining 18 (90%) male samples had normal levels. Similar to this, only 17 (85%) of the 20 female samples had normal levels, while 3 (15%) of them had increased bilirubin levels.

DISCUSSION

The number of diabetic patients is increasing at an alarming rate in developing countries that is approaching epidemic proportions. We know that diabetes affects the kidney in stages. At the very onset of diabetes, the kidney grows large and the glomerular filtration rate (GFR) becomes supranormal. Most recent basic and clinical research have slanted toward sclerosis and kidney failure that occur many years later. Disturbances in liver function tests (LFT) are well recognized in some diabetic patients, especially in acute metabolic decompensation. However, in diabetic patients, the prevalence of abnormal LFTs results and their relationships to clinical findings and diabetes per se, as well as to pathologic changes in liver structure, are controversial.

Total 40 samples were collected for serum Creatinine analysis after analysis it was observed that the range at the age of 40 to 50 year total 13 samples were collected 9 (70%) were

normal and 4 (30%) were elevated. Similarly at the age of 51 to 60 total 12 samples were collected 9 (75%) were normal and 3 (25%) were elevated. Likewise the next age group which ranges from 61 to 70 total 12 sample were taken in which 3 (25%) were elevated and 9 (75%) were normal. And the age group ranges from 71 to 80 total sample size was 3 in which 2 (67%) were infected and 1 (33%) indicates as normal. Similarly total 40 samples were gathered for urea investigation analysis revealed that the age range between 40 and 50 total 8 (61%) of the 13 samples were normal, and 4 (39%) had elevated levels. Similarly, 12 total samples were taken between the ages of 51 and 60, of which 8 (66%) were normal and 4 (33%) were abnormal. Likewise, 12 samples from the age group of 61 to 70 were obtained in total, of which 4 (33%) had increased levels of urea and 8 (66%) had normal levels. The age range is 71 to 80, and the sample size was three, of whom 2 (66%) were elevated and 1 (33%) was normal.

Shrestha *et al.,* (2008) concluded that 18 out of 103 diabetes samples have high urea level whereas 11 out of 103 had increased creatinine level. In control only one sample had high urea value and two had high creatinine level. There was statistical significant increase in urea level with increased in blood sugar level (Shrestha *et al.,* 2008*).*

Results for ALT 12 (92%) of the 13 samples, representing the age range between 40 and 50, were found to be normal, and 1 (8%) had elevated levels after analysis. Similarly, a total of 12 samples between the ages of 51 and 60 were collected, of which 11 (91%) were considered to be normal and 1 (9%) to be abnormal. Likewise, a total of 12 samples from the 61 – 70 age range were collected, of which 6 (50%) had elevated levels and 6 (50%) had normal levels. The 3 people in the sample, 1 (33%) of whom had elevated levels of ALT and 2 (66%) of whom had normal levels, ranged in age from 71 to 80. 40 samples were collected for ALP. After investigation, 8 (61%) of the 13 samples—representing the 40–50 age range—were revealed to be normal, and 5 (39%) showed abnormal levels. Similar to this, 12 samples overall between the ages of 51 and 60 were extracted; 6 (50%) of them were considered to be normal, while 6 (50%) were considered to be high. Similar to this, 12 samples overall from people aged 61 to 70 were gathered, of which 8 (66%) had increased levels and 4 (33%) had normal levels. 2 (66%) of the 3 sample participants had elevated levels, whereas the other 1 (33%) had normal levels. They ranged in age from 71 to 80. 40 samples were also extracted for bilirubin in which 11 (84%) of the 13 samples—representing the 40–50 age range—were found to be normal after review, and 2 (16%) showed elevated values. Similar to this, a total of 12 samples between the ages of 51 and 60 were collected; all of them were determined to have normal levels of serum bilirubin. Similar to this, a total of 12 samples from patients between the ages of 61 and 70 were collected, of which 10 (83%) contained elevated levels and 2 (17%) had normal levels. The levels of 1 (33%) of the 3 sample participants were increased level of bilirubin, while the levels of the 2 (66%) participants were normal. They were between 71 and 80.

Our study supported by (*Salmela*., *et al* 1984) studied that Liver function tests in the diabetic patients. In 100 subjects (57%) of the whole series of 175 diabetic outpatients, at least one of the LFTs used showed abnormal results, and two tests showed abnormal results in 48 subjects (27%). Abnormal Bilirubin was noted in 13%, AP in 34%, Ast in 35%, Alt in 50%, and gGT in 50% of the diabetic inpatients. Two tests gave abnormal results in 53% of these patients *Salmela*., *et al* (1984).

(Ni, H., *et al* 2012) analyzed that total of 81 confirmed diabetes patients participated in this study. Mean age was 56.99 ± 9.06 years and ranged from 22 years to 75 years. Majority of the patients were Bamar (87.7%) and female represented 67.9%. Mean BMI was 25.84, ranging from 22.5 to 30.5. Most of them (97.5%) had type 2 diabetes while only 2 patients (2.5%) had type 1 diabetes.

90.1% were treated with OHA and 63% had family history of diabetes. Regarding clinical characteristics, only 18.5% of the patients had increased liver size, 23.5% had increased liver echo and 16% had fatty liver. Mean value of ALT and AST were 42.94 and 29.69 respectively. Moreover, in this study, serum bilirubin, ALP, rGT and PT were estimated and mean values were 9.13, 102.04, 23.21 and 11.51 respectively (Ni, H., *et al* 2012).

REFERENCES

Arshad, S., Tahir, S., Tahir, B., Tahir, N., Rasool, T., Munir, S., & Junaid, K. (2017). Risk factors associated with diabetes mellitus in local population of Lahore, Pakistan. *Global Journal of Health Science*, *9*(9), 42.

Ni, H., Soe, H. H. K., & Htet, A. (2012). Determinants of abnormal liver function tests in diabetes patients in Myanmar. *Int J Diabetes Res*, *1*(3), 36-41.

Anwar, S. B., Asif, N., Naqvi, S. A. H., & Malik, S. (2019). Evaluation of multiple risk factors involved in the development of diabetic retinopathy. Pakistan journal of medical sciences, 35(1), 156.

Aslam, F., et al. (2019). "White Sesame Seed Oil Mitigates Blood Glucose Level, Reduces Oxidative Stress, and Improves Biomarkers of Hepatic and Renal Function in Participants with Type 2 Diabetes Mellitus." J Am Coll Nutr 38(3): 235-246.

Thomson, S. C., Vallon, V., & Blantz, R. C. (2004). Kidney function in early diabetes: the tubular hypothesis of glomerular filtration. *American Journal of Physiology-Renal Physiology*.

Koye, D. N., Magliano, D. J., Nelson, R. G., & Pavkov, M. E. (2018). The global epidemiology of diabetes and kidney disease. *Advances in chronic kidney disease*, *25*(2), 121-132.

Jha, J. C., Banal, C., Chow, B. S., Cooper, M. E., & Jandeleit-Dahm, K. (2016). Diabetes and kidney disease: role of oxidative stress. *Antioxidants & redox signaling*, *25*(12), 657-684.

Heerspink, H. J. L., & de Zeeuw, D. (2011). The kidney in type 2 diabetes therapy. *The Review of Diabetic Studies*, *8*(3).

Salmela, P. I., Sotaniemi, E. A., Niemi, M., & Mäentausta, O. (1984). Liver function tests in diabetic patients. *Diabetes care*, *7*(3), 248-254.

Ni, H., Soe, H. H. K., & Htet, A. (2012). Determinants of abnormal liver function tests in diabetes patients in Myanmar. *Int J Diabetes Res*, *1*(3), 36-41.

Harris, E. H. (2005). Elevated liver function tests in type 2 diabetes. Clinical diabetes, 23(3), 115-119.

Ritz, E., Rychlík, I., Locatelli, F., & Halimi, S. (1999). End-stage renal failure in type 2 diabetes: a medical catastrophe of worldwide dimensions. *American journal of kidney diseases*, *34*(5), 795-808.

Nosadini, R., Velussi, M., Brocco, E., Bruseghin, M., Abaterusso, C., Saller, A., ... & Fioretto, P. (2000). Course of renal function in type 2 diabetic patients with abnormalities of albumin excretion rate. *Diabetes*, *49*(3), 476-484.

Persson, F., Rossing, P., Reinhard, H., Juhl, T., Stehouwer, C. D., Schalkwijk, C., ... & Parving, H. H. (2009). Renal effects of aliskiren compared with and in combination with irbesartan in patients with type 2 diabetes, hypertension, and albuminuria. *Diabetes care*, *32*(10), 1873-1879.

Targher, G., Lonardo, A., & Byrne, C. D. (2018). Nonalcoholic fatty liver disease and chronic vascular complications of diabetes mellitus. *Nature reviews endocrinology*, *14*(2), 99-114.

Targher, G., & Byrne, C. D. (2013). Nonalcoholic fatty liver disease: a novel cardiometabolic risk factor for type 2 diabetes and its complications. *The Journal of Clinical Endocrinology & Metabolism*, *98*(2), 483-495.

Mandal, A., Bhattarai, B., Kafle, P., Khalid, M., Jonnadula, S. K., Lamicchane, J., ... & Jonnadula, S. (2018). Elevated liver enzymes in patients with type 2 diabetes mellitus and non-alcoholic fatty liver disease. Cureus, 10(11).

Mohamed, J., Nafizah, A. N., Zariyantey, A. H., & Budin, S. (2016). Mechanisms of diabetes-induced liver damage: the role of oxidative stress and inflammation. *Sultan qaboos university medical journal*, *16*(2), e132.

Shrestha, S., Gyawali, P., Shrestha, R., Poudel, B., & Sigdel, M. (2008). Serum urea and creatinine in diabetic and non-diabetic subjects. Journal *of Nepal Association for Medical Laboratory Sciences P, 9(1), 11-12.*

Salmela, P. I., Sotaniemi, E. A., Niemi, M., & Mäentausta, O. (1984). Liver function tests in diabetic patients. *Diabetes care, 7(3), 248-254.*

Ni, H., Soe, H. H. K., & Htet, A. (2012). Determinants of abnormal liver function tests in diabetes patients in Myanmar. Int J Diabetes Res, 1(3), 36-41.