DISCUSSION

The results of the present study have been discussed under the following headings:

5.1 Thyroid related tests
5.2 Prevalence of thyroid dysfunction
5.3 Lipid abnormalities in diabetes
5.4 Renal profile in diabetes
5.5 Liver abnormalities in diabetes
5.6 Correlation between HbA\textsubscript{1C} and thyroid function, lipid profile, renal and liver function tests.

5.1 Thyroid related tests

The mean serum FT\textsubscript{3} (p=<.0001), FT\textsubscript{4} (p=<.0001), T\textsubscript{3} (p=<.0001), T\textsubscript{4} (p=<.0001) were significantly lower in diabetic compared to non-diabetic control subjects, while the mean TSH (p=<.0001) level were significantly higher in diabetic compared to non-diabetic subjects (Table 4.7). Similar results were found when compared the type 1 and type 2 subjects separately with non-diabetic control subjects where the mean values of serum FT\textsubscript{3}, FT\textsubscript{4}, T\textsubscript{3}, T\textsubscript{4} were all significantly lower (p=<.001) and TSH significantly higher (p=<.0001) compared to control subjects (Table 4.10 and 4.13).

These are in agreement with the number of reports on TSH level in type 1 and type 2 diabetes mellitus and many of them have recorded elevated TSH level (Gray \textit{et al.}, 1981; Perros \textit{et al.}, 1995; Flatau \textit{et al.}, 2000; Ditta \textit{et al.}, 2001)

According to Perros \textit{et al} 1995, the overall prevalence of thyroid disease in Scotland was found to be 13.4%, with prevalence of 31.4% in type 1 diabetic females, and lowest in type 2 diabetic males (6.9%).

The Explanation for this finding is that the thyroid hormones, triiodothyroine and tetraiodothyroine are insulin antagonists that also potentiate the action of insulin
Discussion

indirectly (Johnson, 2002). In diabetic patients, the nocturnal TSH peak is blunted and TSH response to TRH is impaired (Gursoy, 1999) which is responsible for the occurrence of low thyroid hormones level in diabetics.

Suzuki et al. (1994) attributed the abnormal thyroid hormones levels found in diabetes to the presence of thyroid hormones binding inhibitor (THBI), an inhibitor of extra thyroidal conversion enzyme (5’-deiodinase) of T₄ to T₃ and dysfunction of the hypothalamus-pituitary-thyroid axis. These situations way prevail in diabetics & would be aggravated in poorly controlled diabetics. Stress, which is associated with diabetes, may also cause changes in the hypothalamus anterior pituitary axis in these diabetics. It appears that the presence of subclinical hypothyroidism and hypothyroidism may results from hypothalamus-hypophyseal- thyroid-axis disorders as suggested by Celani et al. (1994).

There was no significant difference in the level of FT₃ (p=0.668), FT₄ (p=1.00), T₃ (p=0.706), T₄ (p=0.501) and TSH (p=0.080) when the results are compared in (Table 4.16) the diabetic male with diabetic females although the level of serum TSH in diabetic female (6.9 ± 5.81 µIU/ml) is higher as compared to diabetic male (4.9 ± 5.10 µIU/ml).

Also there was no significant difference in the level of serum FT₃ (p=0.2314), FT₄ (p=0.324), T₃ (p=0.2433), T₄ (p=0.1224) and TSH (p=0.5061) when type 1 diabetes were compared with the type 2 diabetes mellitus (Table 4.19).

On comparing (Table 4.25) the level of thyroid hormones in diabetic subjects having abnormal thyroid profile with diabetic subjects having normal thyroid profile, insignificant results were obtained for FT₃ (p=0.739), FT₄ (p=0.6519), T₃ (p=0.2858), T₄ (p=0.861), but the TSH (p = <0.0001) value is significantly increased in diabetic patients having abnormal thyroid profile compared to diabetic patients having normal thyroid profile.

Moreover when diabetic subjects having abnormal thyroid profile compared with non-diabetics subjects having abnormal thyroid profile (Table 4.28), then contrary to the expectation no significant results were obtained for serum levels of FT₃ (p=0.6433), FT₄ (p=0.6301), T₃ (p=0.7144), T₄ (p=0.6091) & TSH (p=0.3715). Possible explanation for this finding was that in non-diabetic
Discussion

subjects having abnormal thyroid profile, there were cases of only hypothyroidism where as in diabetic subjects having abnormal profile there were cases of both hypothyroidism and hyperthyroidism.

Since type 1 and type 2 diabetes have different etiology, the subjects with thyroid dysfunction in both the groups were compared to look for any difference in the levels of thyroid hormones. It was observed that there was no significant difference in the levels of FT$\text{_3}$ (\(p=0.2177\)), FT$\text{_4}$ (\(p=0.2824\)), T$\text{_3}$ (\(p=0.2518\)), T$\text{_4}$ (\(p=0.192\)) \& TSH (\(p=0.2862\)) when type 1 diabetes subjects having abnormal thyroid function were compared with type 2 diabetes subjects having abnormal thyroid function (Table 4.31).

5.2 Prevalence of thyroid dysfunction

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Subjects Group</th>
<th>Prevalance of thyroid dysfunction in diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Celani et al.</td>
<td>1994</td>
<td>T2DM</td>
<td>31.4%</td>
</tr>
<tr>
<td>Smithson</td>
<td>1998</td>
<td>DM</td>
<td>10.8%</td>
</tr>
<tr>
<td>Nobre et al.</td>
<td>2002</td>
<td>T2DM</td>
<td>12.7%</td>
</tr>
<tr>
<td>Bal et al.</td>
<td>2003</td>
<td>DM</td>
<td>40%</td>
</tr>
<tr>
<td>Rajan et al.</td>
<td>2003</td>
<td>T2DM</td>
<td>15%</td>
</tr>
<tr>
<td>Shomon</td>
<td>2003</td>
<td>T1DM</td>
<td>60%</td>
</tr>
<tr>
<td>Radaideh et al.</td>
<td>2004</td>
<td>T2DM</td>
<td>12.5%</td>
</tr>
<tr>
<td>Pimenta et al.</td>
<td>2005</td>
<td>DM</td>
<td>51.6%</td>
</tr>
<tr>
<td>Akbar et al.</td>
<td>2006</td>
<td>T2DM</td>
<td>16%</td>
</tr>
<tr>
<td>Udiong et al.</td>
<td>2007</td>
<td>DM</td>
<td>46.5%</td>
</tr>
<tr>
<td>Gonzalez et al.</td>
<td>2007</td>
<td>T1DM</td>
<td>14.2%</td>
</tr>
<tr>
<td>Pasupati et al.</td>
<td>2008</td>
<td>DM</td>
<td>45%</td>
</tr>
<tr>
<td>Al-Wazzan et al.</td>
<td>2010</td>
<td>T2DM</td>
<td>12.9%</td>
</tr>
<tr>
<td>Papazafilopoulou et al.</td>
<td>2010</td>
<td>T2DM</td>
<td>12.3%</td>
</tr>
<tr>
<td>Shaikh et al.</td>
<td>2010</td>
<td>T2DM</td>
<td>46.6%</td>
</tr>
<tr>
<td>Diez et al.</td>
<td>2011</td>
<td>T2DM</td>
<td>32.4%</td>
</tr>
<tr>
<td>Present Study</td>
<td>2011</td>
<td>DM</td>
<td>29%</td>
</tr>
</tbody>
</table>
There are few studies on the prevalence of thyroid dysfunction in diabetes. Some studies have targeted only type 1 diabetes, while other only type 2 diabetes. The prevalence of thyroid dysfunction varies among different population. The present study concluded 29% of diabetic subjects had thyroid dysfunction (Table 4.20). When this study was compared with different studies from different parts of the world the prevalence of thyroid dysfunction among diabetes mellitus varies. Smithson (1998) reported prevalence of 10.8% thyroid dysfunction in DM patient while Udiong et al. (2007) reported that 46.5% of diabetic patient have thyroid disorder but all these studies showed high incidence of thyroid dysfunction among diabetic as compared to control population.

The prevalence of thyroid dysfunction were more in female as compared to male in this study (Table 4.22). These observations are consistent with the other studies (Gray et al., 1981; Smithson, 1998; Pasupati et al., 2008). This finding is probably associated with the higher prevalence of obesity recorded in female diabetics. Insulin, which is used in treating diabetes and is produced in normal quantities or in excess, has been associated with increased anabolic activity.

Among 100 diabetic subject studied, 24% had lower thyroid hormone level and 5% had higher thyroid hormone level (Total 29% had abnormal thyroid hormone level) where as in non-diabetic subjects, only 4% had lower thyroid hormone level (Table 4.22).

This study is being supported by the other studies by Celani et al.(1994), Rajan et al. (2003), Udiong et al. (2007), Al-Wazzen et al. (2010) on thyroid disorder in diabetes showing the high prevalence of hypothyroidism (both clinical and sub clinical hypothyroidism) as compared to hyperthyroidism. The presence of both raised and low levels of thyroid hormones in diabetes in this study may also be due to modified TRH synthesis and release (Suzuki et al., 1994 ) and may depend on the glycemic status of diabetics studied. Glycemic status is influenced by insulin which in known to modulate TRH and TSH (Reush et al., 1992).

The abnormal thyroid hormone level may be out came of various medication the diabetes were receiving for example it is known that insulin an anabolic hormone enhances the level of FT4, while it suppresses the level of T3 by inhibiting hepatic conversion of T4 to T3. On the other hand, some of the oral hypoglycemic agents
such as the phenythioureas are known to suppress the level of FT$_4$ and T$_4$ while causing raised level of TSH (Suzuki et al., 1994). These situations may explain the finding of low or raised thyroid hormones status in diabetic subjects. Nevertheless the situations in these diabetics does not seem to follow the pattern previously recorded in other non thyroid diseases such as liver diseases and Cushing syndrome where low thyroid hormone level were recorded. The thyroid hormones, triiododthyronine and tetraiodothyronine are insulin antagonists that also potentiate the action of insulin indirectly. These factors could be responsible for the occurrence of low thyroid hormones level in some diabetes.

5.3 Lipid abnormalities in diabetes

Diabetic dyslipidemia is a hallmark of metabolic syndrome and is believed to play an important role in the pathogenesis of atherosclerosis (Fontbonne et al., 1989). Thus it has become major cause for higher cardiovascular morbidity and mortality. Cardiovascular diseases (CVD) are the most prevalent cause of death and disability in both developed as well as developing countries (Chaturvedi et al., 2007). South Asians around the globe have the highest rates of coronary artery disease (CAD) (Enas et al., 2007). According to National Commission in Macroeconomics and Health (NCMH), a government of India undertaking, there would be around 62 million patients with CAD by 2015 in India and of these, 23 million would be patients younger than 40 years of age (Indrayan, 2005). CAD is usually due to atherosclerosis of large and medium sized arteries and diabetic dyslipidemia has been found to be one of the most important contributing factor (NCEP, 2001).

The results of the present study showed significantly increased level of cholesterol (p=<.0001), triglycerides (p=<.0001), LDL-cholesterol (p=<.0001) and VLDL-cholesterol (p=<.0001) and significantly decreased value of HDL-cholesterol (p=<.0001) in diabetic subjects compared to non-diabetic control subjects (Table 4.5).

These result are in good agreement with the study by Siraj et al. (2006), Smith and Lall, (2008) and Pasupati et al. (2010). The main cause of the three cardinal features of diabetic dyslipidemia is the increased free fatty-acid release from insulin-resistant fat cells (Taskinen, 2003; Krauss and Siri, 2004; Chahil et al.,
The increased flux of free fatty acids into the liver in the presence of adequate glycogen stores promotes triglyceride production, which in turn stimulates the secretion of apolipoprotein B (ApoB) and VLDL-cholesterol. The impaired ability of insulin to inhibit free fatty-acid release leads to enhanced hepatic VLDL-cholesterol production (Frayn, 2001), which correlates with the degree of hepatic fat accumulation (Adiels et al., 2007).

Hyperinsulinemia is also associated with low HDL-cholesterol levels (Mooradian et al., 2007). The increased number of VLDL-cholesterol particles and increased plasma triglyceride levels decrease the level of HDL-cholesterol and increase the concentration of small dense LDL-cholesterol particles via several processes: VLDL-transported triglyceride is exchanged for HDL transported cholesterol ester through the action of the cholesterol ester transfer protein (CETP), which results in increased amounts of both atherogenic cholesterol-rich VLDL remnant particles and triglyceride-rich, cholesterol-depleted HDL particles. The increased concentration of small dense LDL-cholesterol particles is explained by a similar lipid exchange. Increased levels of VLDL transported triglyceride enable CETP to promote the transfer of triglyceride into LDL in exchange for LDL transported cholesterol ester. The triglyceride-rich LDL undergoes hydrolysis by hepatic lipase or lipoprotein lipase, which results in lipid-depleted small dense LDL particles (Mooradian et al., 2004).

Among the type 1 diabetic subjects (Table 4.8) also mean levels of cholesterol (p=<0.0001), triglycerides (p=<0.0001), LDL-cholesterol (p=<0.0001), VLDL-cholesterol (p=0.024) were significantly higher where as HDL-cholesterol were significantly lower (p=<0.0001) as compared to non-diabetic subjects. The present data was in agreement with some previous reports (Mann et al., 1978; Court et al., 1978; Sosenko et al., 1980; Lopes-Virella et al., 1981; AI Muhtaseb et al., 1992; AI-Naama et al., 2002) although other (Kobbah et al., 1988 and Salzer et al., 1993) have reported total serum cholesterol to be similar in type 1 and control groups. Possible reasons for difference in reports includes case selection criteria, nature of control population, diet of the general population, the duration and severity of diabetes, the degree of glycemic control and variation in laboratory methods.
In subgroup type 2 diabetic subjects (Table 4.11) mean cholesterol ($p=<.0001$), triglycerides ($p=<0.0001$), LDL-cholesterol ($p=<0.0001$), VLDL-cholesterol ($p=<0.0001$) were significantly higher where as HDL-cholesterol was marginally significantly lower ($p=.064$) as compared to non-diabetic subjects. The results are consistent with previous reports by Bello-Sani et al. (2007), Jali et al. (2010) and Gordon et al. (2010).

Diabetic females have significantly higher serum cholesterol ($p=0.0195$), HDL-cholesterol ($p=<0.0001$), LDL-cholesterol ($p=0.0072$) and insignificantly lower triglycerides ($p=0.2147$) and VLDL-cholesterol ($p=0.5181$) as compared to diabetes males (Table 4.14). These results are in agreement with the studies of Seyoum et al. (2003) and Kayode et al. (2010) although Mengesha (2006) did not find any difference among cholesterol, triglycerides, HDL in diabetic males and females.

Type 2 diabetics has significantly higher triglycerides ($p=0.0437$), HDL ($p=0.0101$), VLDL ($p=0.0012$) but cholesterol ($p=0.0719$) and LDL-cholesterol ($p=.2048$) is not significantly higher than type 1 diabetic subjects (Table 4.17). These results are partially consistent with the study of Seyoum et al. (2003), who found cholesterol, triglycerides and LDL-cholesterol were significantly higher in type 2 diabetic patients than in type 1 diabetic patients but HDL-cholesterol were same in both types of diabetic patients.

Diabetic subjects having abnormal thyroid profile have significantly higher serum cholesterol ($p=<0.0001$), triglycerides ($p=<0.0001$), LDL-cholesterol ($p=<0.0001$), VLDL-cholesterol ($p=<0.0001$) and significantly lower HDL-cholesterol ($p=<0.0001$) than diabetic subjects having normal thyroid profile (Table 4.23). This is consistent with the study of Ardekani et al. (2010).

Also there is a significantly higher level in triglycerides ($p=0.0103$), LDL-cholesterol ($p=0.0338$), VLDL-cholesterol ($p=0.0144$) and marginally significant cholesterol levels ($p=0.0601$) and significantly lower levels of HDL ($p=.0002$) in diabetic subjects having abnormal thyroid profile compared to non-diabetic subjects having abnormal thyroid profile (Table 4.26).
There was no significant difference in the levels of cholesterol (p=0.2047), triglycerides (p=0.1738), HDL (p=0.2094), LDL (p=0.1556) and VLDL-cholesterol (p=0.1191) in type 1 and type 2 diabetic mellitus having abnormal thyroid profile (Table 4.29).

5.4 Renal profile in diabetes

Diabetes mellitus is a slow progressive disease characterized by hyperglycemia. Over time, high blood sugar levels damage million of nephrons-tiny filtering units with in each kidney. As a result, kidneys are unable to maintain the fluid and electrolyte homeostasis. Creatinine is filtered by the glomerulus; therefore, serum creatinine level is used as an indirect measure of glomerular filtration. As glomerular filtration rate (GFR) diminishes, there is a rise in plasma concentration of serum creatinine and urea. Furthermore, the rise indicates progression of kidney disease and estimation of serum creatinine has greater prognostic ability compared with urea for predicting the adverse outcomes.

The result of present study shows that diabetic subjects have significantly higher levels of blood urea (p=<0.0001) and serum creatinine (p=<0.0001) as compared to non-diabetic subjects (Table 4.6). The above result corresponds with the finding of Puepet et al. (2003), Mittal et al. (2010) and Pasupati et al. (2010).

Type 1 diabetic subjects does not show any significant higher levels of blood urea (p=0.205) and creatinine (p=0.112) compared to non-diabetic subjects (Table 4.9), however type 2 diabetic subjects shows significant higher level of blood urea (p=<0.0001) & creatinine (p=<0.0001) compared to non-diabetic subjects (Table 4.12).

In the above finding sex was not the determining factor for the diabetes. Blood urea (p=0.1681) and creatinine (p=0.1955) levels does not shows any significant difference between males and females (Table 4.15). This is consistent with the finding of Shrestha et al. (2008).

Also type 2 diabetes subjects shows significant higher levels of blood urea (p=<0.0001) and creatinine (p=<0.0001) compared to type 1 diabetic subjects (Table 4.18). The possible reason for this may be due to the difference in the treatment of diabetes. Type 1 diabetic patients are on insulin whereas type 2
diabetic patients are mostly on oral drugs, which have more deleterious effects on the kidney compared to insulin.

There was a significant difference between the diabetic subjects having abnormal thyroid profile and diabetic subjects having normal thyroid profile (Table 4.24) for the level of blood urea (p=0.0362) and creatinine (p=0.0104). Thus abnormal thyroid profile in diabetic subjects effect renal dysfunction.

The level of blood urea and creatinine were higher in diabetic subjects having abnormal thyroid profile compared to non-diabetic subjects having abnormal thyroid profile (Table 4.27) although the levels are not significant (p=0.2353 and p=0.1313 respectively).

Significant difference was found in the level of blood urea (p=0.0447) and creatinine (p=0.0278) in type 1 diabetes subjects having abnormal thyroid function compared to the level of type 2 diabetic subjects having abnormal thyroid function (Table 4.30).

### 5.5 Liver abnormalities in diabetes

The liver plays a major role in the regulation of carbohydrate metabolism, as it uses glucose as a fuel, it has the capability to store glucose as glycogen and also synthesize glucose from non-carbohydrate sources. This key function of liver makes it vulnerable to diseases in subjects with metabolic disorders, particularly diabetes (Levinthal & Tavill, 1999).

In this study liver enzymes i.e. SGOT (p=0.0007) and SGPT (p=0.0005) are significantly higher in diabetes compared to non diabetes subjects (Table 4.6). Type 1 diabetic subjects did not shows any significant difference in the level of SGOT (p=0.468) and SGPT (p=0.978) compared to non-diabetic subjects (Table 4.9), where type 2 diabetic subjects shows significant higher level of SGOT (p=<0.0001) and SGPT (p=<0.0001) compared to non-diabetic subjects (Table 4.12). This is in agreement with the study of Idris et al. (2011) who found type 2 sudanese diabetic patients have significant higher liver enzymes compared to controls.
In the above finding sex was the determining factor for diabetes for liver enzymes. Diabetic males had higher level of SGOT and SGPT (Table 4.15) and the difference was statistically significant (p=0.0051 and p=0.0007) respectively. Type 2 diabetic subjects had significantly higher level of SGOT (p=0.0088) and SGPT (p=0.0391) compared with type 1 subjects (Table 4.18). These results were consistent with the finding of Salmela et al. (1984) and Idris et al. (2011).

Abnormal thyroid profile in diabetic subjects have no effect on the level of SGOT (p=0.5949) and SGPT (p=0.7952). This means abnormal thyroid profile does not affect the liver enzymes (Table 4.24).

Although the level of SGOT and SGPT were higher in diabetic subjects having abnormal thyroid profile compared with non-diabetic subjects having abnormal thyroid profile, the levels were non-significant (p=0.4094 and p=0.2984) respectively (Table 4.27).

Also there was no significant difference between type 1 (p=0.4431) type 2 (p=0.4798) diabetic subjects have abnormal thyroid function (Table 4.30).

**5.6 Correlation between HbA1C and thyroid function, lipid profile, renal and liver function tests.**

The correlation coefficient ‘r’ measures the degree and nature of the relationship of one variable with the other and also the direction of correlation between two observations.

Correlation may be due to any of the following factors:

1. One variable being the cause of the other
2. Both variables being the result of a common cause. Here the correlation existing between the two variables may be due to their being related to some other variable

Correlation was studied between HbA1C and lipid profile (Cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol and VLDL-cholesterol), thyroid function test (FT3, T3, FT4, T4 and TSH), renal function test (Blood urea, creatinine) and liver function tests (SGOT and SGPT).
Table 4.32 and 4.33 shows a significant positive correlation between cholesterol and HbA1C in both diabetic (p=.002, r=.307) and non diabetic subjects (p=.026, r=.223). As evident from the r values the correlation was much stronger in diabetics as compared to non-diabetics. Similarly (Table 4.34) a significant correlation was observed between triglycerides and HbA1C among diabetes subjects (p=.0001, r=.405) while in non-diabetic subject (p=.093, r=.169) the levels of triglycerides were insignificantly correlated with HbA1C (Table 4.35). In case of diabetic subjects (Table 4.36) insignificant negative correlation (p=.107, r= -.162) was observed between HDL-cholesterol and HbA1c while on the other hand in non diabetic subjects (p=.553, r=.060) non-significant positive correlation was observed (Table 4.37). Here negative correlation represents decrease in levels of HDL-cholesterol with increase in HbA1c values. Though there was significant positive correlation (Table 4.38 and Table 4.39) between LDL-cholesterol and HbA1c in both diabetic (p=.003, r=.290) and non diabetic subjects (p=.043, r=.203) the correlation was much stronger in diabetics as compared to non-diabetics. There was significant positive correlation (Table 4.40) between VLDL-cholesterol and HbA1c in diabetic subjects (p=.000, r=.409) while there was non-significant positive correlation (Table 4.41) between VLDL-cholesterol and HbA1c in non diabetic subjects (p=.125, r=.154).

Similar results have been reported by Mahato et al. (2011) who observed strong significant correlation between HbA1C and total cholesterol, LDL-cholesterol and HDL-cholesterol in type 2 diabetic subjects. Several other studies (Grey et al.,1981; Perez et al., 2000) have reported that patients with poorly controlled diabetes mellitus show high levels of total cholesterol and triglycerides levels with low HDL levels and observed dyslipidaemia is dependent on glycaemic control.

In both diabetic (p=.000, r=.829) and non diabetic (p=.000, r=.735) subjects there was significant positive correlation between FPG and HbA1c (Table 4.42 and Table 4.43) and finds support from a recent study conducted by Ebesunun and Adedipe (2011).

There was insignificant positive correlation between HbA1c and free T₃ (p=.225, r=.122), T₃ (p=.393, r=.086), free T₄ (p=.128, r=.153) and T₄ (p=.235, r=.120) in diabetic subjects (Table 4.44,Table 4.48,Table 4.46 and Table 4.50 respectively) while there was non-significant negative correlation between HbA1c and free T₃
(p=.504, r= -.068), T₃ (p=.288, r= -.107), free T₄ (p=.181, r= -.135) and T₄ (p=.247, r= -.117) in non diabetic subjects (Table 4.45, Table 4.49, Table 4.47 and Table 4.51). There was significant positive correlation (Table 4.52 and Table 4.53 respectively) between TSH and HbA1c in both diabetic (p=.021, r=.230) and non diabetic subjects (p=.028, r=.219). The results are supported by study conducted by Udiong et al. (2007) who reported significant association (r = 0.211, p = .012) between TSH and HbA1C in type 2 diabetics.

There was significant positive correlation (Table 4.54) between SGOT and HbA1c in diabetic subjects (p=.003, r=.295) while there was non-significant positive correlation (Table 4.55) between SGOT and HbA1c in non diabetic subjects (p=.559, r=.059). Similarly there was significant positive correlation (Table 4.56) between SGPT and HbA1c in diabetic subjects (p=.002, r=.304) and non significant positive correlation (Table 4.57) between SGPT and HbA1c (p=.594, r=.054) in non diabetic subjects.

There was significant positive correlation (Table 4.58 and Table 4.59 respectively) between blood urea and HbA1c in diabetic (p=.016, r=.240) and non diabetic subjects (p=.005, r=.278). Similarly there was significant positive correlation (Table 4.60 and Table 4.61 respectively) between creatinine and HbA1c in diabetic (p=.008, r=.262) and non diabetic subjects (p=.004 r=.283).