Type 2 Diabetes Mellitus in Egyptian Diabetics Under Forty Years

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ABSTRACT

AIMS: The aims of our study are to investigate whether type 2 diabetes mellitus (T2DM) occurs only in adults and whether age is a perfect parameter to distinguish type 1 diabetes mellitus (T1DM) from T2DM and to confirm that the disturbance of liver functions in diabetic patients is not due to Hepatitis C virus (HCV) infection. METHOD: We evaluate nine type 2 diabetic patients with age range (13-39 years) in The Medical Unit in the National Research Center, Cairo, Egypt. results: There is a significantly increase (P < 0.05) in fasting and post prandial glucose concentrations in T2DM as compared with control although insulin concentration was normal by treatment. Patients are showing normal levels and non significant value of alanine amino transferase (ALT) enzyme (P = 0.05), a significant increase in Aspartate amino transferase (AST) enzyme (P < 0.05), non significant increase of alkaline phosphatase (ALP) enzyme, total bilirubin and direct bilirubin (P = 0.9, 0.7 and 0.4), respectively. Hepatitis C virus-antibodies (HCV-Abs) consider non significant negative (P = 0.95) as compared with control. conclusions: A higher fasting and post prandial glucose concentrations associate with a younger age at diabetes onset. Higher liver function tests in T2DM expect more accompanied liver diseases. Although, HCV-Abs are negative there are elevations in liver function tests.

Key Words: Type 2 diabetes, hepatitis C virus, age, insulin resistance and liver functions.

INTRODUCTION

Diabetes is a syndrome characterized by disordered metabolism and high blood glucose level, hyperglycemia, resulting from low levels of insulin production by beta cells of the pancreas. The characteristic symptoms are excessive urine production, Polyuria, excessive thirst and increased fluid intake, polydipsia (Tierney et al. 2002).

There are two main types of diabetes mellitus exist, insulin-dependent diabetes mellitus (IDDM) also known as type 1 diabetes (T1DM) and non–insulin dependent diabetes mellitus (NIDDM) also known as type 2 diabetes (T2DM) (WHO 1999).

T1DM and T2DM are not completely distinctive clinically or etiologically and may overlap considerably such as acute presentation or insulin requirement may be present in T2DM and autoimmune phenomena may arise in combination with insulin resistance (Rapaport et al. 2001).

The possible relationship between T1DM and T2DM is a controversial subject. Formally, the two are considered to be etiologically distinct. T1DM is characterized by autoimmune destruction of pancreatic beta cells, resulting in a failure to produce insulin. T2DM is caused by impaired beta cell function and capacity to secrete insulin, coupled to a decline in tissue sensitivity to insulin (Cervin et al. 2008). Type 2 diabetes is generally occurring after age 40 and so, it is known as adult-onset diabetes and it occurs due to insulin resistance or reduced insulin sensitivity (Eberhart et al. 2004).

In United States of America (USA), a dramatic increase in the incidence of T2DM in children has been noted. Glucose toxicity of the pancreas, which occurs with chronic hyperglycemia and impairs insulin secretion, may lead to manifestation of florid diabetic ketoacidosis and requires insulin therapy at diagnosis. For successful long-term management of T2DM is in a child, a program that includes weight reduction must be implemented (Pohl et al. 1998).

Clinical trials aimed at the prevention of overfeeding and the control of weight gain in children may not only prevent T2DM in later life, but may also contribute to a delayed manifestation of T1DM (Buyken et al. 2010).
Additional environmental and physiological stresses such as pregnancy, weight gain, physical inactivity and medications may result in the development of insulin resistance. As the beta-cells fail to compensate for the prevailing insulin resistance, impaired glucose tolerance and diabetes develops. As glucose levels rise, beta-cell function with diminishing sensitivity to glucose and hyperglycemia. The pancreatic islet cell mass is reported to be reduced in size in diabetic patients (Nielsen et al. 2001).

Well-known standards for the diagnosis of T2DM are based on values of fasting blood glucose, random blood glucose and the oral glucose-tolerance test and are identical for adults and children. Normal fasting plasma glucose is <100 mg/dL. Patients with fasting levels between 100 and 125 mg/dL have impaired fasting glucose. Patients with fasting levels ≥126 mg/dL have diabetes. Two elevated readings on 2 separate days are needed to make a diagnosis. A random or “casual” plasma glucose value ≥200 mg/dL is diagnostic of diabetes if the patient has additional symptoms such as polyuria. During an oral glucose-tolerance test, a 2-hour plasma glucose value of <140 mg/dL is considered normal, ≥140 and <200 mg/dL is considered impaired glucose tolerance and ≥200 mg/dL is diagnostic of diabetes (Hannon et al. 2005).

Increasing rates of T2DM among children will have considerable long-term implications for the affected individuals, society and the public health system as a whole (Rosenbloom 2002). Earlier onset of T2DM leads to earlier onset of complications including progressive neuropathy, retinopathy leading to blindness, nephropathy leading to chronic renal failure and atherosclerotic cardiovascular disease leading to stroke, myocardial infarction and (in some cases) sudden death. In addition to their impact on physical well-being, the economic, social and psychological impact of these conditions is enormous (Gortmaker et al. 1993).

Early in the evaluation of a new patient with diabetes mellitus, it is important to distinguish between T1DM and T2DM to optimize therapy. Clinical signs helpful in distinguishing T2DM from T1DM are signs of insulin resistance (hypertension and/or polycystic ovary syndrome). Patients with T2DM frequently have elevated insulin and C-peptide levels. The absence of auto antibodies to insulin and/or glutamic acid decarboxylase is also typical in most (but not all) cases of diabetes that are classified as T2DM (Aguilar-Salinas et al. 2001). Weight loss and/or prevention of weight gain are the best ways to prevent T2DM among children with risk factors for the disease (Barlow and Dietz 1998).

Pre-diabetes is a condition that raises the risk of developing type 2 diabetes, heart disease, stroke and eye disease. People with pre-diabetes are 5-15 times more likely to develop T2DM than people are with normal glucose values (Santaguida et al. 2005).

Insulin resistance, which develops as a result of both genetic and environmental factors, is now widely believed to be the first step in the development of T2DM, cardiovascular disease and other conditions (Wajchenberg 2000). Glucose homeostasis is maintained by insulin secretion, insulin action, hepatic glucose production and cellular glucose uptake (DeFronzo 1988). Insulin sensitivity and insulin secretion are inversely and proportionately related. The lower the insulin sensitivity (i.e., the greater the insulin resistance), the more insulin that is secreted (Krebs and Jacobson 2003).

Because insulin resistance is often associated with T2DM, the first step in assessment is to identify children who would benefit from intervention. Testing has been recommended for children at significant risk for the presence or development of T2DM. These are children who in general are overweight, have a family history of T2DM, have a predisposition based on race/ethnicity and have signs of insulin resistance or conditions associated with insulin resistance. At this time, sufficient data are not available to support the use of the glycosylated hemoglobin (HbA1c) in the diagnosis of diabetes. It is important to remember that even in the face of a normal fasting glucose level, the child may have diabetes or remain at risk for developing T2DM. Children who do not have elevated blood glucose concentrations may exhibit other features of the insulin resistance syndrome, such as obesity, hypertension and high cholesterol and they remain at risk for cardiovascular disease and diabetes (Steinberger and Daniels 2003).

HCV infection may contribute to the development of diabetes. Glucose intolerance is observed more often in patients with HCV infection compared with control (Fraser et al. 1996). Type 2 diabetes is more common in women with than in those without HCV infection. This association and its potential mechanisms may have clinical implications. Investigation into the mechanisms linking HCV infection to the expression of T2DM may also help to define processes that promote the development of T2DM in susceptible individuals (Wilson 2004).

Patients with T2DM seem more likely to have a range of liver diseases and patients with both liver diseases and diabetes are at risk of severe liver disease. This has obvious implications for the clinical management. Awareness of type 2 diabetes as a significant risk factor for liver injury may improve diagnosis and interventions to minimize the progression of chronic liver disease (Hickman and Macdonald 2007).
**Statistical analysis:**
All obtained data were analyzed using the student's t-test and the analysis of variance test (ANOVA) applications of the Graph Pad InStat soft ware version 3.01 (Graph Pad Soft ware, Inc). Results described as Mean ± Standard Deviation.

**SUBJECTS AND METHODS**

**Patients' sera:**
Nine human sera samples were selected from 82 randomly selected type 2 diabetic patients (9/82; 10.9%) (Data did not show). They were (6 males / 3 females) with age range from (13 - 39) years (mean 27.1 ± SD 9.7) according to Eberhart et al. (2004). This was kindly provided by Dr. Amany Abdel Ghany at the Medical Unit in the National Research Center, Cairo, Egypt.

Molecular, immunological and biochemical diagnosis were achieved by collecting 10 ml venous fasting blood samples in the morning after 6-8 hours overnight fast. Seven ml were left to clot in plastic Hoffman' tubes to use for biochemical analysis for liver function and immunological determination of insulin, 0.5 ml was collected in Fluoride tubes and mixed well to use for measuring of glucose level. Also, 2.5 ml were collected in gel tubes and mixed well to use for molecular detection of HCV by PCR technique. Then all tubes were put in centrifuge at 4000 r.p.m. for 15 minutes to obtain on separated serum. Samples were analyzed immediately or stored at -20 till use.

- **Control human sera:**
Ten venous fasting blood samples were collected from ten control subjects with no history of diabetes disease. They were (4 males / 6 females) with age (mean 22.4 ± SD 8.75) years. These sera show normal values in liver function tests, blood glucose test and were negative in HCV-Abs ELISA test.

**Determination of fasting and post prandial glucose levels in human sera:**
Reagents were obtained from BioSystems Reagents and Instruments (Barcelona, Spain REF. NO. 11504). Colorimetric method was used to estimate serum glucose using glucose oxidase and peroxidase (Trinder 1969) at wavelength 520 nm.

- **Liver function tests:**

  - **Determination of serum alanine aminotransferase (ALT):**
  Reagents were obtained from BioSystems Reagents and Instruments (Barcelona, Spain REF. NO. 11533) and serum ALT was estimated according to Gella (1985) kinetic method at wave length 340 nm.

  - **Determination of serum aspartate aminotransferase (AST):**
  Reagents were obtained from BioSystems Reagents and Instruments (Barcelona, Spain REF. NO. 11531) and serum AST was estimated according to Gella (1985) kinetic method at 340 nm.

**Normal range:** For male ≤ 37 and female ≤ 32 U/L.

- **Determination of serum alkaline phosphatase (ALP):**
Reagents were obtained from BioSystems Reagents and Instruments (Barcelona, Spain REF. NO. 11592) and serum ALP was estimated by kinetic method according to (IFCC 1983) at wave length 405 nm.

**Normal range:** For male and female ≤ 80 U/L.

- **Determination of total and direct bilirubin:**
Serum bilirubin was determined by the colorimetric method (Walters 1970) at wave length 535 nm and the reagents were obtained from Bio-diagnostic (Egypt CAT. NO. BR 1110).

**Normal range:** For total bilirubin ≤ 1mg/dl and for direct bilirubin ≤ 0.25 mg/dl.

**Quantitative measurement of fasting insulin in human sera using enzyme linked immunosorbent assay (ELISA):**
Reagents were provided from (DRG International Inc., USA REF. NO. EIA-2935) and the test was performed according to manufacturer’s instructions. The insulin ELISA kit is a solid phase based on the sandwich principle. An aliquot of patient sample containing endogenous insulin was incubated in the coated well with enzyme conjugate, which was an anti-insulin antibody conjugated with Biotin. After 30 minutes the unbound conjugate was washed off by washing solution. During the second incubation step for another 30 minutes, streptavidin peroxidase enzyme complex binds to the biotin-anti-insulin antibody. The amount of bound horseradish peroxidase (HRP) complex is proportional to the concentration of insulin in the sample. Having added the substrate solution, the intensity of colour developed is proportional to the concentration of insulin in the sample.

**Qualitative analysis of HCV-antibodies using ELISA in human sera:**
The kit (SV HCV ELISA 3.0 REF. NO. 204055) was obtained from (Standard diagnostic Inc., Korea) and the test was performed according to manufacturer’s instructions. The microplate wells were pre-coated with recombinant HCV antigens (core, NS3, NS4 and NS5). During first incubation, anti-HCV in patient' serum was bound to the recombinant HCV antigens. Then all unbound materials were removed by washing. The anti-human IgG peroxidase enzyme conjugate was bound to anti-HCV.
Then all unbound materials were removed by washing also. The residual enzyme activity will be directly proportional to the anti-HCV concentration in patient's serum and evidenced by incubating the solid-phase with a substrate solution in a substrate buffer. Absorbance was measured with spectrophotometer (Unicam UV 300, Germany) at wavelength 450 nm with reference wavelength at 620 nm.

Examination of HCV-RNA in patients' sera by RT-PCR:
Viral RNA extraction kit was obtained from Qiagen (QIAamp, Qiagen, Hilden, Germany) Cat. No. 52904 was according to manufacturer’s instructions. Diethylpyrocarbonate (DEPC) was obtained from (Sigma, Deisenhofen, Germany). Amplification of Oligonucleotide primers was done using GoTaq Flexi DNA polymerase according to (Bahgat et al. 2009). Primer pair amplified partial sequence of a molecular weight 302 bp region:

**Sense primer:** 5' CAC TCC CCT GTG AGG AAC T 3'

**Anti-sense primer:** 5' TGG ATG CAC GGT CTA CGA GAC CTC 3'.

Agarose gel electrophoresis of PCR product was carried out according to Maniatis et al. (1982). Analysis of PCR product was done according to Wicks et al. (1986).

The PCR product bands were visualized on the gel documentation system (Biometra, Goettingen, Germany). The bands were analyzed in comparison to the 100 bp DNA marker using the Lab image analyzer software version 2.7.0.

RESULTS

Characterization of control subjects:
A) Biochemical determination of fasting, post prandial glucose and immunological measurement of fasting insulin concentration using ELISA in normal control sera: Biochemical examination of fasting, post prandial blood glucose and immunological examination of fasting insulin level (Figure 1) in control subjects show normal levels (84.2±4.78 mg/dl), (147.1±19.95 mg/dl) and (11.4±4.37 IU/ml), respectively.

B) Biochemical determination of liver enzymes and immunological determination of HCV-Abs using ELISA in sera from nine type 2 diabetic patients. Table (2) shows the levels of liver enzymes (ALT, AST, ALP, total and direct bilirubin) and HCV-Abs concentration in sera from nine type 2 diabetic patients with an age 13-40 years. ALT is specific for liver but AST is less specific.

Patients are showing normal levels of ALT enzyme (34.8±23.3, P = 0.05) except cases no. 5, 9 that give expression about liver inflammation of those patients, a significant increase in AST enzyme (44.8±33.4, P < 0.05). Patients no. 1, 3, 9 had increased AST levels that may be due to liver inflammation also although HCV-Abs is negative. There are a non significant increase of ALP enzyme (69.4±40.9, P = 0.9), total bilirubin (0.61±0.56, P = 0.7) and direct bilirubin (0.18±0.22, P = 0.4) in sera from patients. HCV-Abs considered negative (0.25±0.09, P = 0.95) as compared with control.

C) Detection of HCV-RNA by RT-PCR:
As shown in Fig. (2), the five normal subjects were negative for RT-PCR detection of HCV-RNA which confirms the absence of HCV infection.

Characterization of nine type 2 diabetic patients with age range (13-40 years)
A) Biochemical determination of fasting, post prandial glucose levels and immunological measurement of fasting insulin in sera from type 2 diabetic patients. Figures (3a&b) showed the biochemical examination of fasting, post prandial blood glucose and immunological examination of fasting insulin level in nine type 2 diabetic patients. There is a significant increasing of fasting glucose (185.3±112.7, P < 0.05) as compared with normal values of fasting glucose in control subjects (84.2±4.78 mg/dl). Post prandial glucose shows a significant increasing (310.2±112.8, P < 0.05) as compared with post prandial glucose normal values in control subjects (147.1±19.95 mg/dl) (Figure 3a). Patients no. 3, 6, 9 had FBS <100mg/dl and the PPS ranged from 250-500mg/dl due to treatment tablets taken by them. There is no significant change (9.9±5.2, P = 0.56) in fasting insulin level as compared with normal values of control subjects (11.4±4.37 IU/ml) due to treatment using tablets (Figure 3b).

B) Biochemical determination of liver enzymes and immunological determination of HCV-Abs using ELISA in sera from nine type 2 diabetic patients. Table (2) shows the levels of liver enzymes (ALT, AST, ALP, total and direct bilirubin) and HCV-Abs concentration in sera from nine type 2 diabetic patients with an age 13-40 years. ALT is specific for liver but AST is less specific.

Patients are showing normal levels of ALT enzyme (34.8±23.3, P = 0.05) except cases no. 5, 9 that give expression about liver inflammation of those patients, a significant increase in AST enzyme (44.8±33.4, P < 0.05). Patients no. 1, 3, 9 had increased AST levels that may be due to liver inflammation also although HCV-Abs is negative. There are a non significant increase of ALP enzyme (69.4±40.9, P = 0.9), total bilirubin (0.61±0.56, P = 0.7) and direct bilirubin (0.18±0.22, P = 0.4) in sera from patients. HCV-Abs considered negative (0.25±0.09, P = 0.95) as compared with control.

C) Detection of HCV-RNA by RT-PCR:
Figure (4) is confirming absence of HCV infection in type 2 diabetic patients' sera by RT-PCR and HCV-Abs normal concentration using ELISA (Table 2).
Table 1: Liver functions and HCV-antibodies in sera of control subjects (n = 10).

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (year)</th>
<th>Sex</th>
<th>ALT (U/L)</th>
<th>AST (U/L)</th>
<th>ALP (U/L)</th>
<th>Bilirubin (mg/dl)</th>
<th>HCV-Abs conc.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Total</td>
<td>Direct</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>22</td>
<td>Female</td>
<td>25</td>
<td>18</td>
<td>54</td>
<td>0.275</td>
<td>0.020</td>
</tr>
<tr>
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<td>33</td>
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<td>38</td>
<td>10</td>
<td>73</td>
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</tr>
<tr>
<td>3</td>
<td>18</td>
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<td>30</td>
<td>32</td>
<td>65</td>
<td>0.490</td>
<td>0.210</td>
</tr>
<tr>
<td>4</td>
<td>33</td>
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<td>12</td>
<td>12</td>
<td>75</td>
<td>0.21</td>
<td>0.168</td>
</tr>
<tr>
<td>5</td>
<td>26</td>
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<td>14</td>
<td>15</td>
<td>47</td>
<td>0.196</td>
<td>0.098</td>
</tr>
<tr>
<td>6</td>
<td>12</td>
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<td>30</td>
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<td>7</td>
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<td>58</td>
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<tr>
<td>8</td>
<td>12</td>
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<td>24</td>
<td>66</td>
<td>0.186</td>
<td>0.018</td>
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<tr>
<td>9</td>
<td>38</td>
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<td>74</td>
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<tr>
<td>10</td>
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<td>18</td>
<td>26</td>
<td>73</td>
<td>0.756</td>
<td>0.322</td>
</tr>
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</table>

Table 2: Liver function tests and HCV-antibodies concentration in sera from type 2 diabetic patients (n = 9).

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (year)</th>
<th>Sex</th>
<th>ALT (U/L)</th>
<th>AST (U/L)</th>
<th>ALP (U/L)</th>
<th>Bilirubin (mg/dl)</th>
<th>HCV-Abs conc.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td>Total</td>
<td>Direct</td>
<td></td>
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<td>136</td>
<td>0.336</td>
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<td>82</td>
<td>53</td>
<td>0.21</td>
<td>0.112</td>
</tr>
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</table>

Figure 1: Biochemical characterization of normal control subjects. (a) Concentration of fasting and post prandial glucose in sera from normal subjects, (b) Concentration of fasting insulin in sera from normal subjects.

Figure 2: RT-PCR for ten normal subjects (lanes 1-5). M: marker, +ve: positive control at 302 bp length.
DISCUSSION

Type 1 diabetes mellitus commonly occurs in childhood or adolescence, although the rising prevalence of T2DM in these age groups is now being seen worldwide (Bogdanović 2008).

Type 2 diabetes mellitus has long been considered a disease of adults, in whom it is the most prevalent form of diabetes, about (90%) and is associated with increased risk of cardiovascular disease morbidity and mortality (Vinicore 1994). During the past 10 years, however, an increasing frequency in the occurrence of type 2 diabetes mellitus has been reported in children.

The young diabetic patients are usually diagnosed as type 1 diabetic patients which is most often occurs in childhood (WHO 1999) but there wasn't any one of our patients treated with insulin injections which oppose with Hirsch (1999) who reported that type 1 diabetics usually inject with insulin. So, aged criteria of diagnosing diabetes into type 1 (less than 40 years) or type 2 (more than 40 years) might be incorrect. T2DM occurred in young persons less than 40 years also. So, while most type 2 diabetics are older than 40, the condition can develop at any age.

Type 2 diabetes can remain undetected for many years and the diagnosis is often made from associated complications or incidentally through an abnormal blood or urine glucose test (Strader and Steeff 2001).

Type 2 diabetic patients have normal or increased plasma insulin levels. Insulin resistance results in the development of diabetes and is commonly found in unaffected first-degree relatives. The morbidity of the disorder relates both to the severity of hyperglycemia and the metabolic insulin resistance itself (Hunter and Garvey 1998).

Our results showed a significant increase of fasting and post prandial glucose concentration in type 2 diabetic patients while there was non significant increase in fasting insulin as compared with control due to treatment tablets taken by all patients such as metformin, sulfonyl ureas (glimepiride, glyburide and glipizide) and meglitinides (repaglinide and nateglinide) (Hannon 2005).

There is a high prevalence of elevated serum aminotransferases among children with T2DM in agreement with Nadeau et al. (2005) who reported that there is a high prevalence of elevated serum aminotransferases among children in their study unrelated to age, body mass index, glycemic control, blood lipids or diabetic therapy.

As in the adult population, T2DM in children and youth occurs as a result of insulin resistance coupled with relative beta-cell failure. T2DM has been shown as more common in patients with hepatitis C virus infection (HCV). There is increased prevalence of DM in patients with HCV. HCV precedes the development of DM by a decade (Amarapurkar and Patel 2008).

In our type 2 diabetic patients, there was a significant increase in AST enzyme while there was no significant
increase in ALT, ALP, total and direct bilirubin as compared with control. HCV-Abs level was also negative.

CONCLUSION

Type 2 diabetes occurs in children as well as adults. Age is not enough parameter to distinguish type 1 and type 2 diabetic patients. Insulin resistance is contributing to T2DM which occurs also in children rather than adults. Higher levels of liver function tests may expect more liver diseases. Diabetes mellitus is one of the most common chronic diseases known to increase the susceptibility of the affected individuals to viral infections.

REFERENCES


