Original Article

Study of life style habits on risk of type 2 diabetes

Rajiv Pathak, Ashima Pathak
Department of Physiology, Faculty of Medicine, SEGi University, Kota Damansara, Malaysia, 'Department of Biotechnology, GGDSD College, Sector 32, Chandigarh, India

Abstract

Background: Diabetes mellitus has become one of the great epidemics of our time. Aim: Characterized by derangement of carbohydrate, protein and fat metabolism. Diabetic patients may have some other habits like drinking, smoking, lack of physical activity. In the present study, we have tried to study the effect of all these habits on lipid profile and antioxidative enzymes, i.e., catalase and superoxide dismutase. Materials and Methods: Different kits and standard biochemical methods were used to estimate all these parameters. Results: Diabetics as well as diabetic individuals who were engaged in drinking, smoking and regular physical exercise showed a significant rise in glucose levels compared to normal subjects. Similarly, cholesterol, triglyceride and high-density lipoprotein cholesterol levels and activity of catalase were found to be increased in all diabetic subjects, but that of superoxide dismutase decreased as compared to normal subjects. In all cases, exercise has a beneficial effect. Furthermore, females were more prone to destructive effects of diabetes than males. Conclusion: We can conclude that smoking and drinking by diabetic subjects further deteriorates the effects of diabetes, while regular physical exercise has beneficial effects.

Key words: Antioxidative enzymes, drinking, diabetes mellitus, lipid profile, physical activity, smoking

Introduction

Diabetes mellitus is a metabolic disorder characterized by hyperglycemia and is one of the leading causes of death. Non-insulin dependent diabetes mellitus is partially of genetic etiology,[1] but it also strongly influenced by environmental factors, and life-style of the patient. Oxidative stress and overproduction of reactive oxygen species (ROS), which occur during diabetes, play important roles in cardiovascular morbidity and mortality.[2] All cells utilize molecular oxygen for metabolic purposes and are at the risk of being damaged by oxygen-derived free radicals and lipid peroxidation products. Complementary antioxidants provide defense against this oxidative injury. The antioxidant defense mechanisms range from low molecular-weight compounds (glutathione, tocopherols, selenium, and vitamin E) to complex enzymatic systems (superoxide dismutase, glutathione peroxidase and catalase). A disturbance of the cellular pro-oxidant/antioxidant balance in favor of the former may denote an oxidative stress.[3]

Diabetes is a complex disease where the carbohydrate and fat metabolism is impaired.[4] It is well known that diabetes is associated with an increased incidence of macrovascular complications, including coronary artery disease (CAD), and cerebrovascular and peripheral disease.[5] The elevated triglyceride (TG) levels may be caused by medical conditions such as diabetes, hypothyroidism, kidney disease, or liver disease. Dietary causes of elevated TG levels may include obesity and high intakes of fat, alcohol, and concentrated sweets. Several studies have indicated the high prevalence of hyperlipidemia, which may have harmful effect on the diabetic patients and, therefore, there is a need of monitoring serum lipid concentration in diabetic patients. Therefore, assessment of various lipid fractions in cases of diabetes mellitus may be of some help in the prognosis of patients and in preventing the possibilities of complication or secondary disorders.

Factors such as age, sex and certain aspects of life-style,
including diet, level of physical activity, and level of diabetes control also affect lipid profile. People who have type 2 diabetes tend to have high levels of triglycerides and low levels of high-density lipoprotein (HDL) and similar low-density lipoprotein (LDL) levels than people who do not have diabetes. Both sexes show an increase in lipoprotein levels as they age.

There are independent risks of diabetes with smoking and alcohol consumption. Epidemiological studies of alcohol intake and risk of type 2 diabetes have pronounced conflicting results. Several studies have indicated that moderate drinkers had the lowest risk for diabetes, and non-drinkers and heavy drinkers had a higher risk. Excessive alcohol intake may contribute to excess intake and obesity, induction of pancreatitis, disturbance of carbohydrate metabolism, and impairment of liver function. Smoking has also been identified as a risk factor for resistance, which can lead to diabetics.

The importance of sedentary life-style as a risk factor for diabetes and of the protective effects of physical activity in the maintenance of diabetes has also been studied. Regular physical exercise enhances the antioxidant defense system and protects against exercise induced free radical damage. On the other hand, intense exercise in untrained individuals overwhelms defenses resulting in increased free radical damage. Thus, the “weekend warrior” who is predominantly sedentary during the week but engages in vigorous bouts of exercise during the weekend may be doing more harm than good.

**Materials and Methods**

This retrospective study was conducted on subjects with/without type 2 diabetes mellitus. The patients (150) were matched for age, gender, glucose levels, and diabetes duration with 30 patients in each group. Patient selection was made during the period of 3 months. The subjects with blood glucose levels ≤110 mg/dl and who were neither on any medication nor having any other disease were taken as healthy controls. Type 2 diabetes mellitus was considered to be present if the fasting blood glucose was ≥126 mg/dl and the patient was using glucose lowering medication.

Subjects with any other disease, including diabetic complications and also those under antioxidant medication or lipid lowering drugs were excluded from the study. The diabetics were allowed their regular life styles.

All procedures followed were approved by the institutional ethical committee and were carried according to the Helsinki Declaration of 1975, as revised in 2000 and with the written consent and adequate understanding by all the volunteers after explaining the nature and purpose of the study. The fasting blood samples were procured from the known normal healthy (non-diabetic control) and diabetic subjects from the diabetic clinic.

All the investigations were performed in the serum of diabetic and control subjects. Following groups were made based on the life-style of diabetic subjects with the help of the questionnaire provided to them: Control; diabetes alone; diabetics involved in smoking; diabetics engaged in drinking; and diabetics doing exercise.

Blood samples procured were allowed to stand at room temperature for 30 min, centrifuged at 1500 rpm (revolutions per minute) and serum was separated carefully from the pellets.

**Biochemical estimations**

Cholesterol, triglyceride, and HDL-cholesterol (HDL-C) were estimated using the kits obtained from Reckon Diagnostics Pvt. Ltd. Baroda, India. The chemicals used in other parameters were obtained from Sisco Research Laboratories (SRL) Pvt. Ltd., Mumbai.

Glucose was estimated by the method of Trinder et al. Triglyceride was estimated by the method of Cole et al. Cholesterol was estimated by the method of Allain et al. HDL-C was estimated by the method of Tietz et al. Catalase was estimated in the kinetic mode at 240 nm in double beam ultraviolet spectrophotometer (Shimadzu) by the method of Luck. The activity of superoxide dismutase was estimated according to the method of Kono.

**Statistical analysis**

The comparison between control and various groups was done using ANOVA. P value <0.05 was considered significant.

**Results**

Glucose levels were found to be elevated significantly in all the groups as compared to normal subjects. However, the increase in glucose levels was less pronounced in case of exercising diabetic subjects [Table 1]. When the glucose levels of males and females were compared, females were found to have much higher glucose levels (P < 0.05) than males [Table 2]. Cholesterol levels were found to be raised in all the groups, but this increase was significant (P < 0.001) only in the diabetic subjects and in the diabetics who were also engaged in drinking (P < 0.05) as compared to the normal ones. However, in all these cases, the cholesterol levels were found to be in the normal range. Similar trend was noticed in the levels of triglycerides and LDL-cholesterol (LDL-C) in all the groups [Table 3].
However, HDL-C levels were found to be decreased significantly in all the groups when compared to the normal individuals. When the lipid profile of the male and female diabetic subjects were compared, it was again noticed that females were more prone to the damage due to diabetes than the male subjects [Table 4]. Activity of catalase was found to have significantly increased in the diabetic patients \( (P < 0.01) \), in the diabetic subjects doing exercise \( (P < 0.001) \) and diabetics on drinking \( (P < 0.01) \) as compared to the healthy subjects. On the other hand, in the diabetics who are smoking also, the activity of catalase was found to be decreased, in comparison with the normal individuals [Table 5].

The activity of superoxide dismutase was found to be increased in patients with exercise but decreased in all other groups. However, the decrease was found to be significant \( (P < 0.01) \) only in case of diabetes with smoking patients [Table 5]. Females were found to have high catalase and superoxide dismutase activity than in the males, but this rise was not statistically significant in both cases [Table 6].

**Discussion**

It is a well-established fact that diabetes is a risk factor for cardiovascular disease.\(^{[5]}\) Cardiovascular complications are the leading cause of morbidity associated with diabetes. Since diabetes mellitus has been associated with lipid abnormalities,\(^{[4]}\) it has been customary to utilize the measurement of blood glucose, triglycerides, total cholesterol, HDL-C and LDL-C in profiling the risk of diabetes. Impairment of insulin secretion leads to an excessive and prolonged rise in glucose concentration. As observed in our study, diabetic smokers also had higher blood-glucose levels as compared to the normal individuals which may be because smoking has been known to stimulate the release of the counter regulatory hormones and consequently, cause temporal elevation in blood-glucose levels.\(^{[18]}\) Alcohol intake by diabetic individuals also showed a significant elevation in the blood-glucose levels in their serum, and this rise was even more than that observed in case of only diabetic individuals as compared to the normal ones. However, contradictory studies also showed that there is no dose–response relation between alcohol intake and glucose levels.\(^{[19]}\) Glucose levels were also found to be elevated in the exercising diabetic persons, but this increase was less prominent than was observed in other groups. This shows that regular physical activity can prevent or postpone the onset of type 2 diabetes. It was also confirmed in the present study that females are more prone to developing diabetes than the males.

There is growing evidence that excess generation of highly reactive free radicals, largely due to hyperglycemia, causes

| Table 1: Glucose values in different diabetic groups |
|-----------|-----------------|-----------------|
| Group     | Glucose levels (mg/dl) |
|-----------|-----------------|
| Normal    | 103.0±6.50      |
| Diabetic  | 187.1±34.76***  |
| Exercise  | 150.0±16.90     |
| Drinking  | 218.3±50.90***  |
| Smoking   | 190.2±35.16**   |

\( ^{*}P<0.05; ^{**}P<0.01; ^{***}P<0.001 \) compared to normal

| Table 2: Effect of gender on glucose levels in diabetic subjects |
|-----------------|-----------------|-----------------|
| Group           | Glucose levels (mg/dl) |
|-----------------|-----------------|
| Male            | 213.6±8.09      |
| Female          | 267.0±35.16*    |

\( ^{*}P<0.05 \)

| Table 3: Effect of drinking, smoking and exercise on serum lipid profile in diabetic subjects |
|-----------------|-----------------|-----------------|
| Group           | Cholesterol     | Triglyceride     | HDL-C            | LDL-C            |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| Normal          | 154.3±36.78     | 139.9±11.77     | 48.3±10.85      | 83.7±18.09      |
| Diabetic        | 184.5±34.62***  | 181.0±39.39***  | 29.4±10.03      | 105.3±32.05     |
| Exercise        | 61.5±17.35      | 151.8±20.64     | 35.6±7.54*      | 85.4±9.18***    |
| Drinking        | 179.0±24.42a    | 209.2±27.70***  | 33.6±13.36      | 99.2±13.33      |
| Smoking         | 165.2±19.21     | 168.7±18.43*    | 28.4±11.25      | 105.2±31.26     |
|                | F value         | 11.50           | 7.54            | 3.90            | 8.59            |
|                | P value         | <0.01           | <0.01           | <0.05           | <0.05           |

All values in mg/dl; HDL-C: High-density lipoprotein cholesterol; LDL-C: Low density lipoprotein cholesterol; \( ^{a}P<0.05; ^{**}P<0.01; ^{***}P<0.001 \) compared to normal

| Table 4: Effect of gender on lipid profile in diabetic subjects |
|-----------------|-----------------|-----------------|
| Group           | Cholesterol     | Triglyceride     | HDL-C            | LDL-C            |
|-----------------|-----------------|-----------------|-----------------|-----------------|
| Male            | 180.0±11.08     | 198.0±11.89     | 46.8±3.64       | 83.7±20.40      |
| Female          | 227.0±22.111*** | 219.0±7.34      | 57.4±4.91       | 110.3±30.05***  |

All values in mg/dl; \( ^{***}P<0.001 \)

| Table 5: Effect of drinking, smoking and exercise on the activity of catalase and superoxide dismutase in the serum of diabetic subjects |
|-----------------|-----------------|-----------------|
| Group           | Catalase        | Superoxide dismutase |
|-----------------|-----------------|-----------------|
| Normal          | 2.83±1.47       | 0.33±0.18       |
| Diabetic        | 4.22±1.09***    | 0.25±0.08       |
| Exercise        | 5.25±1.19***    | 0.36±0.13       |
| Drinking        | 4.99±0.64***    | 0.23±0.01       |
| Smoking         | 2.17±1.47       | 0.19±0.12**    |
|                | F value         | 21.62           | 3.13            |
|                | P value         | <0.001          | <0.05           |

\( ^{***}P<0.01; ^{***}P<0.001 \) compared to normal

| Table 6: Effect of gender on the activity of catalase and superoxide dismutase in the serum of diabetic subjects |
|-----------------|-----------------|-----------------|
| Group           | Catalase        | Superoxide dismutase |
|-----------------|-----------------|-----------------|
| Male            | 3.05±1.64       | 0.27±0.12       |
| Female          | 4.12±1.05       | 0.35±0.20       |
|                | F value         | 1.18            | 1.02            |
|                | P value         | NS              | NS              |

NS: Not significant
Hyperglycemia also results in increased production of the ROS within numerous biochemical pathways that can lead to an increase in antioxidative enzymes. This state of excess generation of free radicals, observed both in type 1 (insulin-dependent) and type 2 (non-insulin-dependent) diabetes causes oxidative stress. This oxidative stress exacerbates the development and progress of diabetes and its complications. Recently, a hypothesis was proposed that suggested that all the above processes involved in the elevation of oxidative stress are a consequence of superoxide by mitochondrial respiratory chain during hyperglycemia. In the present study, significantly higher activity of the antioxidant enzymes, i.e., catalase and decrease in the activity of superoxide dismutase were found in diabetic patients as compared to the normal individuals who were taken as control. Superoxide dismutase activity is undoubtedly important to the regulation of oxidative stress in diabetes. However, there is a variation as to the status of this enzyme in the diabetic state. Some studies have reported decreased superoxide dismutase activity, while others have shown increases or no change in the enzyme activity.

This finding suggests that both walking and more physical activity are associated with reductions in risk of diabetes and activity may slow the initiation and progression of type 2 diabetes and its cardiovascular sequelae via favorable effects on body weight, insulin sensitivity, glycemic control, blood pressure, lipid profile, endothelial function, and inflammatory defense systems. However, more studies are needed to determine the intensity, frequency, and duration of exercise that is more effective in prevention of diabetes. Thus, although our observational study falls short in unraveling cause–effect relationships, but it is tempting to speculate that for adequate treatment of diabetes, increased emphasis on antioxidative enzymes and lipid profile, either pharmacologically or through lifestyle modifications, may constitute a valuable means to attenuate diabetes risk in apparently healthy population.

Acknowledgment

We are thankful to all members of the Department of Biotechnology, GGDSD College for providing the laboratory facilities. We take the responsibility of the integrity of the work as a whole from inception to published article and should be designated as “guarantors.”

References

1. Jarrett RJ. Epidemiology and public health aspects of non-insulin-dependent diabetes mellitus. Epidemiol Rev 1989;11:151-71.
2. Guzik TJ, Harrison DG. Vascular NADPH oxides as drug targets for novel antioxidant strategies. Drug Discov Today 2006;11:524-33.
3. Vertua S, Anguri A, Manfredini S. The antioxidant and pro-antioxidants network: An overview. Curr Pharm Des 2004;10:1677-94.
4. Alteme E, Vendemis G, Chico D. Increased lipid peroxidation in type II poorly control diabetic patients. Diabetes Metab 1991;18:264-71.
5. Laakso M. Hyperglycemia and cardiovascular disease in type 2 diabetes. Diabetes 1999;48:937-42.
6. Gillies CI, Abrams KR, Lambert PC, Cooper NJ, Sutton AJ, Hsu RT, et al. Pharmacological and lifestyle interventions to prevent or delay type 2 diabetes in people with impaired glucose tolerance: Systematic review and meta-analysis. BMJ 2007;334:299.
7. Bazzano LA, Seluka M, Liu S. Prevention of type 2 diabetes by diet and lifestyle modification. J Am Coll Nutr 2005;24:310-9.
8. Perry JJ, Wannamethee SG, Shaper AG. Prospective study of serum gamma-glutamyltransferase and risk of NIDDM. Diabetes Care 2003;26:874-6.
9. Clarkson PM. Antioxidants and physical performance. Clin Rev Food Sci Nutr 1995;35:131-41.
10. Goldfarb AH. Antioxidants: Role of supplementation to prevent exercise-induced oxidative stress. Med Sci Sports Exerc 1993;25:232-6.
11. Trinder P. Determination of glucose in blood using glucose oxidase with an alternative oxygen acceptor. Ann Clin Biochem 1969;6:24.
12. Cole TG, Klotsch SG, McNamara J. Measurement of triglyceride concentration. In: Handbook of Lipoprotein Testing. Washington: AACC Press; 1997. p. 115-26.
14. Allain CC, Poon LS, Chan CS, Richmond W, Fu PC. Enzymatic determination of total serum cholesterol. Clin Chem 1974;20:470-5.
15. Burtis CA, Ashwood ER, editors. Tietz Textbook of Clinical Chemistry. 3rd ed. Philadelphia: WB Saunders Company; 1999. p. 1799-1845.
16. Luck H. Catalase. In: Bergmeyer HO, editor. Methods of Enzymatic Analysis. New York: Academic Press; 1971. p. 885-93.
17. Kono Y. Generation of superoxide radical during autoxidation of hydroxylamine and an assay for superoxide dismutase. Arch Biochem Biophys 1978;186:189-95.
18. Sandberg H, Roman L, Zavadnick J, Kupers N. The effect of smoking on serum somatotropin, immunoreactive insulin and blood glucose levels of young adult males. J Pharmacol Exp Ther 1973;184:787-91.
19. Kato I, Kiyohara Y, Kubo M, Tanizaki Y, Arima H, Iwamoto H, et al. Insulin-mediated effects of alcohol intake on serum lipid levels in a general population: The Hisayama Study. J Clin Epidemiol 2003;56:196-204.
20. Ballantyne D, White C, Strevens EA, Lawrie TD, Lorimer AR, Manderson WG, et al. Lipoprotein concentrations in untreated adult onset diabetes mellitus and the relationship of the fasting plasma triglyceride concentration to insulin secretion. Clin Chim Acta 1977;80:323-9.
21. Kobayashi J, Maruyama T, Watanabe H, Kudoh A, Tateishi S, Sasaki T, et al. Gender differences in the effect of type 2 diabetes on serum lipids, pre-heparin plasma lipoprotein lipase mass and other metabolic parameters in Japanese population. Diabetes Res Clin Pract 2003;62:39-45.
22. Craig WY, Palomaki GE, Johnson AM, Haddow JE. Cigarette smoking-associated changes in blood lipid and lipoprotein levels in the 8- to 19-year-old age group: A meta-analysis. Pediatrics 1990;85:155-8.
23. Manson JE, Spelsberg T. Risk modification in the diabetic patient. In: Manson JE, Ridker PM, Gaziano JM, Hennekens CH, editors. Prevention of Myocardial Infarction. New York: Oxford University Press; 1996. p. 241-73.
24. Kedziora-Kornatowska K, Szram S, Kornatowski T, Szadukis-Szadurski L, Kedziora J, Bartosz G. Effect of vitamin E and vitamin C supplementation on antioxidative state and renal glomerular basement membrane thickness in diabetic kidney. Nephron Exp Nephrol 2003;95:e134-43.
25. Ahmed FN, Naqui FN, Shafiq F. Lipid peroxidation and serum antioxidant enzymes in patients with type 2 diabetes mellitus. Ann N Y Acad Sci 2006;1084:481-9.
26. Maritim AC, Sanders RA, Watkins JB 3rd. Effects of alpha-lipoic acid on biomarkers of oxidative stress in streptozotocin-induced diabetic rats. J Nutr Biochem 2003;14:288-94.

How to cite this article: Pathak R, Pathak A. Study of life style habits on risk of type 2 diabetes. Int J App Basic Med Res 2012;2:92-6.

Source of Support: Department of Biotechnology, GGDSD College, Sector 32, Chandigarh. Conflict of Interest: None declared.

Dispatch and return notification by E-mail

The journal now sends email notification to its members on dispatch of a print issue. The notification is sent to those members who have provided their email address to the association/journal office. The email alerts you about an outdated address and return of issue due to incomplete/incorrect address.

If you wish to receive such email notification, please send your email along with the membership number and full mailing address to the editorial office by email.