

CHANGE IN SERUM CHOLINESTERASE ACTIVITY IN JAMAICAN DIABETICS

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This study investigates the alteration of serum cholinesterase levels in diabetics and its possible relationship to blood glucose, insulin, triglyceride, and cholesterol levels.

Fourteen phasic insulin-dependent diabetes mellitus patients were compared with 10 insulin-dependent diabetes mellitus, 10 noninsulin-dependent diabetes mellitus, and 10 normal controls. Each group was matched for age, sex, body mass index, and duration of diabetes. Mean age was 56.7 ± 2.5 years; mean body mass index, 24.0 ± 0.8 kg/m²; and mean duration of diabetes, 14.2 ± 2.2 years. Serum acetylcholinesterase, insulin, triglyceride, and cholesterol levels as well as fasting blood sugar were all assayed using standard techniques. Results suggest an associated increase of serum acetylcholinesterase with triglyceride levels in diabetics and may point to a possible association between increased serum acetylcholinesterase and vascular complications in Jamaican diabetics. (*J Natl Med Assoc.* 1992;84:853-855.)

Key words • diabetes mellitus • serum cholinesterase
• acetylcholinesterase • triglyceride • Jamaica

The biological role of serum cholinesterase and erythrocyte acetylcholinesterase has not been clearly established. However, it has been reported earlier that cholinesterase may be involved in lipoprotein metabo-

lism, as it has been demonstrated in patients with hyperlipidemia,^{1,2} obesity, and diabetes.³ It also has been reported⁴ that erythrocyte acetylcholinesterase levels are significantly decreased in insulin-dependent diabetes and have a negative correlation with fasting blood glucose levels.

This study investigates the possible relationship between serum acetylcholinesterase and the aberrations in metabolism observed in the diabetic state.

METHODS AND MATERIALS

The population studied consisted of outpatients attending the Diabetic Clinic at the University Hospital of the West Indies, Kingston, Jamaica. The sample consisted of 14 nonobese phasic insulin-dependent diabetes mellitus (PIDDM), 10 insulin-dependent diabetes mellitus (IDDM), 10 noninsulin-dependent diabetes mellitus (NIDDM), and 10 normal controls. The groups were matched for age, weight and sex, and duration of diabetes.

The diagnosis of diabetes was based on fasting plasma glucose and oral glucose tolerance tests using World Health Organization criteria.⁵ Plasma glucose was estimated using the automated ferricyanide method,⁶ plasma insulin was assayed using the Coat-a-Count (Diagnostic Products Corporation, Los Angeles, California) insulin diagnostic kit assay, and serum cholesterol and triglyceride levels were determined using enzymatic methods.⁷ Acetylcholinesterase activity was measured using acetylthiocholine as a substrate as described by Ellman et al.⁸

RESULTS

Table 1 summarizes the clinical details of the various groups. There was no significant difference in body mass index, age, duration of diabetes (for diabetic groups), and male:female ratio for the four groups. The

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TABLE 1. CLINICAL DETAILS AND CHARACTERISTICS OF DIABETIC GROUPS AND NORMAL CONTROLS

Group	n	Mean ± Standard Error						
		Sex		Body Mass Index (kg/m ²)	Age (Years)	Duration of Diabetes (Years)	Fasting Blood Sugar (mmol/L)	Insulin Plasma (pmol/L)
		Male	Female					
IDDM	10	4	6	23.2 ± 1.9	54.0 ± 5.4	14.7 ± 2.7	14.5 ± 2.8	6.6 ± 1.8
NIDDM	10	5	5	24.1 ± 0.9	59.0 ± 2.9	11.8 ± 1.4	9.7 ± 1.9	9.4 ± 2.2
PIDDM	14	6	8	24.7 ± 0.9	57.0 ± 3.2	16.0 ± 1.3	11.8 ± 1.4	8.6 ± 1.7
Normals	10	6	4	22.7 ± 0.7	47.0 ± 4.5	—	4.6 ± 0.3	20.8 ± 1.3

Abbreviations: IDDM = insulin-dependent diabetes mellitus, NIDDM = noninsulin-dependent diabetes mellitus, and PIDDM = phasic insulin-dependent diabetes mellitus.

TABLE 2. PLASMA ACETYLCHOLINESTERASE, TRIGLYCERIDE, AND CHOLESTEROL CONCENTRATIONS FOR DIABETIC GROUPS AND NORMAL CONTROLS

Groups	n	Mean ± Standard Error (IU/mL)		
		Acetylcholinesterase	Triglycerides	Cholesterol
IDDM	10	2.2 ± 0.1	2.0 ± 0.9	5.2 ± 0.5
NIDDM	10	2.4 ± 0.4	1.7 ± 0.2	5.6 ± 0.5
PIDDM	14	2.3 ± 0.1	1.8 ± 0.3	5.5 ± 0.2
Normal	10	1.7 ± 0.06	0.23 to 1.47*	3.6 to 5.2*

Abbreviations: IDDM = insulin-dependent diabetes mellitus, NIDDM = noninsulin-dependent diabetes mellitus, and PIDDM = phasic insulin-dependent diabetes mellitus.

*Range.

blood sugar levels were all significantly elevated in the diabetic groups ($P < .05$) over the normal controls, while the plasma insulin levels were significantly lower ($P < .05$) in all diabetics when compared with normal controls.

Table 2 lists the plasma acetylcholinesterase concentration in the three groups of diabetics as well as in normal controls. Analysis of variance showed that there was no difference in serum concentration of acetylcholinesterase between the three diabetic groups. The mean acetylcholinesterase value for the three diabetic groups (2.3 ± 0.06 IU/mL) was significantly elevated over the normal mean value (1.7 ± 0.06 IU/mL; $P < .01$).

The mean triglyceride levels for the diabetic patients in all three groups were elevated above the normal range (Table 2). Analysis of variance within the diabetic groups showed that there was no real difference in their triglyceride levels ($P = .693$). Postanalysis calculations showed that the entire diabetic group had a mean of 1.8 ± 0.3 mmol/L, which was significantly elevated over the normal upper limit ($P < .05$). In addition, the serum cholesterol concentration of the three diabetic groups were all in the upper normal range (3.9 to 5.2 mmol/L) (Table 2).

DISCUSSION

The elevated serum acetylcholinesterase concentration ($P < .01$) in diabetics showed a similar pattern to serum triglyceride levels (Table 2). The biological function of this enzyme is still an enigma.⁹ Several reports in the literature present evidence suggesting that cholinesterase may be involved in lipoprotein metabolism.^{1,3} A direct relation was observed between enzyme activity and low-density lipoprotein concentrations. Inhibition of the enzyme was associated with a decrease in serum total cholesterol and an increase in high-density lipoprotein concentration.¹⁰

The hypertriglyceridemia seen in the diabetics in this study is a common metabolic aberration of diabetes.¹¹ The mechanism of hyperlipidemia in diabetes mellitus differs according to the type of diabetes. In IDDM, there is reduced lipoprotein lipase activity as a direct consequence of the insulin deficiency. Also in NIDDM, with hyperinsulinemia, hepatic triglyceride synthesis is stimulated.¹² The mechanism in PIDDM remains to be elucidated. Even in the most simple experimental models of diabetes, the elevation of serum triglyceride may be the result of increased endogenous production of very low-density lipoproteins and chylomicra, or a

defective removal of circulating triglyceride, or a combination of both these mechanisms.¹¹

The serum cholesterol of the diabetics in all three groups (Table 2) was in the upper limit of the normal range, which is a common finding in diabetes mellitus.¹³ Also, it must be noted that triglycerides are the metabolic precursors of low-density lipoproteins, which are the main vehicle of cholesterol transport.¹⁴

Lipid concentrations seem to form the basis for vascular complications seen in diabetics.¹⁵ If this is true of our diabetics, then the lipid-related complications in our patients should be more or less the same since they were matched for age, sex, body mass index, and duration of diabetes (Table 1). This was reported previously in one of the matched preliminary studies conducted at the University Hospital of the West Indies.¹⁶

CONCLUSION

The findings of this study show a relationship between the serum concentrations of acetylcholinesterase and triglyceride in Jamaican diabetics. Because triglyceride and atherogenicity in diabetes is well established, we can extrapolate our results to a possible association between increased serum acetylcholinesterase and vascular complications in diabetes.

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