Original Article

Cognitive Function and Control of Type 2 Diabetes Mellitus in Young Adults

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Abstract

Background: Type 2 diabetes mellitus (T2DM) has been associated with impairment of cognitive function. Studies show a strong negative correlation between the levels of glycosylated hemoglobin and cognitive function in adult patients above the mean age of 60 years. In healthy adults, age-related cognitive impairment is mostly reported after the age of 60 years, hence the decline in cognitive function can be a part of normal aging without diabetes. Since the majority of patients with diabetes are between the ages of 40 and 59 years, it is crucial to ascertain whether the levels of glycosylated hemoglobin negatively correlate with the levels of cognitive function scores in adult patients of age 60 years or younger, similar to the way it correlates in patients older than 60 years of age, or not. **Aims:** We observed the relationship between the levels of glycosylated hemoglobin and the levels of cognitive function in patients of age 60 years or younger with T2DM. **Materials and Methods:** Eighty-two patients with T2DM underwent cognitive assessment testing by using a Modified Mini-Mental State Examination (3MS), and their cognitive function scores were correlated with their glycosylated hemoglobin levels, durations of diabetes, and levels of education. **Results:** Cognitive impairment was observed in 19.5% of the studied patients. We found a weakly negative relationship between the glycosylated hemoglobin level and cognitive function score (r = -0.292), a moderately negative relationship between the duration of diabetes and cognitive function score (r = -0.303), and a weakly positive relationship between the level of education and cognitive function score (r = 0.277). **Conclusion:** Cognitive impairment affects one-fifth of the patients of age 60 years or younger with T2DM. It is weakly negatively related to the glycosylated hemoglobin level, moderately negatively related to the duration of diabetes, and weakly positively related to the level of education.

Keywords: Cognitive function, cognitive impairment, diabetes, duration of diabetes, glycosylated hemoglobin, level of education, type 2 diabetes mellitus (T2DM)

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Introduction

A global epidemic, diabetes afflicts nearly 382 million people worldwide, a number that will increase by 55% and is predicted to reach more than 592 million by the year 2035.[1] Three-fourths of all patients with diabetes live in China, India, and the USA.[1] While type 2 diabetes mellitus (T2DM) has been associated with coronary artery disease, hypertension, renal disease, and obesity both as cause and effect, many studies have also raised concerns about the long-term consequences of poor glycemic control on the impairment of cognitive function.[2-8] It is important to mention that these studies were conducted in patients of all ages, and mostly in patients above the mean age of 60 years. Although the exact pathophysiology of cognitive impairment in T2DM is unclear, hyperglycemia, vascular disease, hypoglycemia, insulin resistance, amyloidosis, concomitant hypertension, and depression play significant roles.[9,10] In healthy adults, age-related cognitive impairment may begin during early adulthood, but is mostly reported after the age of 60 years.[11] The prevalence of mild cognitive impairment in healthy

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adults above the age of 65 years is 10-20%,[12,13] thus it is safe to say that decline in cognitive function can be expected after the age of 60 years as a part of normal aging even without T2DM. On the other hand, less than 4% of the healthy adults in the USA who are of age 65 years or younger suffer from dementia,[14] and there are no data available on the prevalence of cognitive impairment in this population. Since a majority of the patients with diabetes are between the ages of 40 and 59 years,[1] it is crucial to ascertain whether poor control of T2DM affects cognitive function in this population of younger adults similar to the older adults, or not. We designed this study to observe the relationship between the level of glycosylated hemoglobin as a marker of control of T2DM and the level of cognitive function in young adult patients of age 60 years or younger who presented at our primary care office.

Materials and Methods

Study selection

This study was a prospective, single-arm assessment study of cognitive function in patients with T2DM who were managed either by diet alone or by diet and drug therapy. Patient enrollment started in May 2013 and was completed in November 2014. The study was reviewed and approved by the Institutional Review Board of the Cooper Health System, Camden, New Jersey, USA. Adult patients between the ages of 18 and 60 years who presented at our internal medicine office with T2DM were approached by the study physicians for possible enrollment during their scheduled routine office visits. All patients received a description of the study and they were informed about the purpose, risks, benefits, alternatives, and required follow-ups. Informed consent was obtained from each participating patient. Cognitive function was assessed once by the established assessment tool (Modified Mini-Mental State Examination, 3MS),[15] which is questionnaire-based. The study physicians also reviewed the electronic medical records of the subjects for data collection. The inclusion criteria were: English language-speaking adult patients between the ages of 18 and 60 years, who had T2DM for more than 3 months. The exclusion criteria ruled out: patients older than 60 years; patients with established diagnosis of dementia due to any cause; patients who were not self-administering medications; patients who could not communicate in English; patients with an advanced comorbid medical condition that could have affected cognitive function, such as advanced neurological condition (e.g., cerebrovascular accident, Parkinson's disease, multiple sclerosis), advanced cardiac condition with poor performance state [e.g., New York Heart Association (NYHA) III or IV congestive heart failure, cardiomyopathy with left ventricular ejection fraction of less than 40%], advanced pulmonary disorder (e.g., chronic obstructive or restrictive airway disease requiring ambulatory oxygen therapy, end-stage renal disease on hemodialysis, end-stage liver disease (e.g., cirrhosis), hematological disorders leading to severe anemia (hemoglobin less than 9.0 g/dL), advanced uncontrolled rheumatological disorder (e.g., rheumatoid arthritis, systemic lupus erythematosus, osteoarthritis), advanced multisystem disorder (e.g., sarcoidosis), developmental disorders, mental retardation, and uncontrolled psychiatric disorders. We excluded patients aged over 60 years due to the possibility of the presence of cognitive impairment irrespective of T2DM, which can be expected in this population based on the studies that have shown a strong association between age greater than 60 years and a decline in cognitive function in healthy individuals.[11-13]

Data collection

After obtaining informed consent, we administered the 3MS to the participating patients. We collected the following data for each patient: age, gender, race, highest education level, duration of T2DM, associated medical condition, current medications to control T2DM, blood pressure (mmHg), hemoglobin (Hb)A_{1c} (%), microalbuminuria (mcg/mL), low-density lipoprotein cholesterol (LDL-C mg/dL), triglyceride (mg/dL), and 3MS score.

Statistical analysis

We entered the patient data in a Microsoft Excel (2013, Redmond, Washington) spreadsheet, and analyzed them using SPSS (Statistical Package for the Social Science, version 15.01, IBM, Armonk, New York). We analyzed the mean 3MS score based on age, gender, and duration of T2DM-based subgroups, and correlated the 3MS score with HbA_{1c} and other parameters. A total of 82 patients were planned for sequential sampling in order to achieve the largest sample that would provide 80% power, 5% alpha error, and a medium effect size of 30% to the study hypotheses. The patients' education level and duration of diabetes were considered in the statistical analysis through correlation with the scores on the 3MS scale. An independent *t*-test was used to compare the mean 3MS score to gender and the one-way analysis of variance (ANOVA) test was used to compare 3MS scores for each age category. We used Pearson's correlation to observe the correlation between the 3MS score with HbA_{1c}, age groups, and race. The point biserial correlation was used to correlate the 3MS score with the age groups, gender, and comorbid conditions.

Results

A total of 82 patients with T2DM participated in the study. A cognitive test (3MS) was administered to all of the participating patients. Normal cognitive function

was defined as a 3MS score between 79 and 100, while a score under 79 was defined as cognitive impairment. Cognitive impairment was observed in 19.5% patients.

Baseline characteristics

All patients with T2DM were between the ages of 26 and 60 years. The mean age of the patients in the normal cognitive function group was 49 years; 34.8% were non-Hispanic Caucasian, 34.8% were African-American, 10.7% were Hispanic, and 17% were of other races, mainly Asian. The mean age of the patients in the cognitive impairment group was 52 years; 18.8% were non-Hispanic Caucasian, 50.0% were African-American, 25.0% were Hispanic, and 6.2% were of other races, mainly Asian [Table 1]. Half of the

Table 1: Baseline characteristics

Variable	All patients (*n* = 82)	Patients with T2DM and normal cognitive function (*n* = 66)	Patients with T2DM and cognitive impairment (*n* = 16)
Age			
Years, mean (SD)	50 (9)	49 (9)	52 (9)
Gender			
Male, *n* (%)	44 (53.7)	36 (54.5)	8 (50.0)
Female, *n* (%)	38 (46.3)	30 (45.5)	8 (50.0)
Race			
Non-Hispanic Caucasian, *n* (%)	26 (31.7)	23 (34.8)	3 (18.8)
African-American, *n* (%)	31 (37.8)	23 (34.8)	8 (50.0)
Hispanic, *n* (%)	11 (13.4)	7 (10.7)	4 (25.0)
Other, *n* (%)	14 (17.1)	13 (19.7)	1 (6.2)
Education			
Beyond high school, *n* (%)	39 (47.6)	35 (53.0)	4 (25.0)
High school or less, *n* (%)	43 (52.4)	31 (47.0)	12 (75.0)
Duration of T2DM			
Less than 5 years, *n* (%)	14 (17.1)	13 (19.7)	1 (6.3)
5-10 years, *n* (%)	45 (54.9)	39 (59.1)	6 (37.5)
More than 10 years, *n* (%)	23 (28.0)	14 (21.2)	9 (56.2)
Management			
Diet alone, *n* (%)	5 (6.1)	5 (7.6)	0 (0.0)
Diet + OHA, *n* (%)	37 (45.1)	34 (51.5)	3 (18.8)
Diet + Insulin, *n* (%)	17 (20.7)	12 (18.2)	5 (31.2)
Diet + OHA + Insulin, *n* (%)	23 (28.1)	15 (22.7)	8 (50.0)
Comorbid medical diagnoses			
Coronary artery disease, *n* (%)	8 (9.8)	6 (9.1)	2 (12.5)
Hypertension, *n* (%)	61 (74.4)	49 (74.2)	12 (75.0)
Hypothyroidism, *n* (%)	14 (17.1)	12 (18.2)	2 (12.5)
Depression, *n* (%)	17 (20.7)	13 (19.7)	4 (25.0)
Congestive heart failure, *n* (%)	2 (2.4)	2 (3.0)	0 (0.0)
COPD, *n* (%)	4 (4.9)	2 (3.0)	2 (12.5)
Anemia, *n* (%)	4 (4.9)	3 (4.5)	1 (6.3)
Arthritis, *n* (%)	13 (15.9)	9 (13.6)	4 (25.0)
CKD, *n* (%)	5 (6.1)	2 (3.0)	3 (18.8)
Peripheral neuropathy, *n* (%)	25 (30.5)	15 (22.7)	10 (62.5)
Retinopathy, *n* (%)	8 (9.8)	3 (4.5)	5 (31.2)
Malignancy, *n* (%)	2 (2.4)	1 (1.5)	1 (6.3)
Vitals			
Systolic BP (mmHg), mean (SD)	128.26 (14.00)	127.08 (13.44)	133.14 (15.61)
Diastolic BP (mmHg), mean (SD)	78.96 (8.69)	78.44 (8.76)	81.3 (8.29)
Laboratory values			
Microalbuminuria (mcg/mL), median (IQR)	7.55 (2.08 to 30.75)	8.10 (1.95 to 37.20)	5.40 (2.40 to 21.05)
LDL (mg/dL), mean (SD)	95.20 (37.53)	116.31 (40.77)	90.08 (35.15)
Triglyceride (mg/dL), mean (SD)	175.06 (120.76)	174.64 (127.14)	176.81 (93.28)

SD = Standard deviation, T2DM = Type 2 diabetes mellitus, OHA = Oral hypoglycemic agent, COPD = Chronic obstructive pulmonary disease, CKD = Chronic kidney disease; BP = Blood pressure, LDL = Low-density lipoprotein

patients in the cognitive impairment group were African-American. Overall, the majority of the patients (56.1%) were in the age range of 51-60 years. The majority of the patients in the normal cognitive function group (53.0%) and in the cognitive impairment group (68.6%) were in the same age group, of 51-60 years [Figure 1]. While males represented a little more than half (54.5%) of the patients in the normal cognitive function group, males and females were represented equally in the cognitive impairment group. Patients in the cognitive impairment group had a higher prevalence of certain comorbid conditions, such as depression, chronic obstructive pulmonary disease (COPD), arthritis, chronic kidney disease (CKD), peripheral neuropathy, and retinopathy [Table 1]. We found no significant difference in blood pressure, level of microalbuminuria, LDL-C, and triglyceride between the normal cognitive function group and the cognitive impairment group [Table 1].

Level of education and cognitive function

There was a weakly positive relationship between the level of education and cognitive function scores ($r = 0.277$). Although cognitive impairment was observed more in patients who had an education of high school or under, the difference between this group and the patients who had an education beyond high school was not significant ($P = 0.044$).

Duration of T2DM and cognitive function

There was a moderately negative relationship between the duration of T2DM and cognitive function ($r = -0.303$) [Figure 2]. Although there was no relationship between the duration of T2DM and normal cognitive function ($r = -0.157$), in the cognitive impairment group we observed that more than half of the patients had T2DM for more than 10 years' duration, and there was an incremental pattern of cognitive impairment with the increase in the duration of T2DM, establishing a strongly negative relationship ($r = -0.407$) [Figure 3].

Management of T2DM and cognitive function

About half (51.5%) of the patients in the normal cognitive function group were managed by diet and one or more oral hypoglycemic drugs followed by 22.7% patients who were managed by diet, oral hypoglycemic drug(s) and insulin. On the other hand, half (50%) of the patients in the cognitive impairment group were managed by diet, oral hypoglycemic drug(s), and insulin, followed by about one-third (31.2%) of the patients who were managed by diet and insulin [Table 1].

Glycemic control and cognitive function

Cognitive impairment was observed in 11.6% of the patients who had optimal glycemic control (HbA_{1C} under 7%), and 30.2% who did not have optimal glycemic control (HbA_{1C} 7% or greater) ($P < 0.001$) [Figure 4]. Overall, we found a weakly negative relationship between the glycosylated hemoglobin level and the cognitive function score ($r = -0.292$). There was no relationship between the glycosylated hemoglobin level and the cognitive function score in patients with optimal glycemic control ($r = -0.049$), while there was a weakly negative relationship between the glycosylated hemoglobin level and the cognitive function score in patients who did not have optimal glycemic control ($r = -0.273$). There was no relationship between the ascending ranges of poor glycemic control and cognitive impairment [Figures 5 and 6]. There was no relationship between the cognitive function and age, degree of microalbuminuria, LDL-C level, and triglyceride level.

Discussion

Our study demonstrated two major observations: first, a weakly negative relationship between the glycosylated hemoglobin level and the cognitive function, and second, a 19.5% prevalence of cognitive impairment. Our observation of a weakly negative relationship stands markedly different from the observations of the majority of the studies that have shown a very strong negative

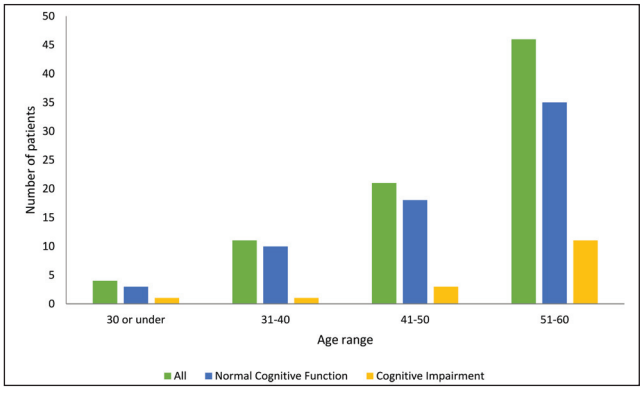

Figure 1: Age distribution

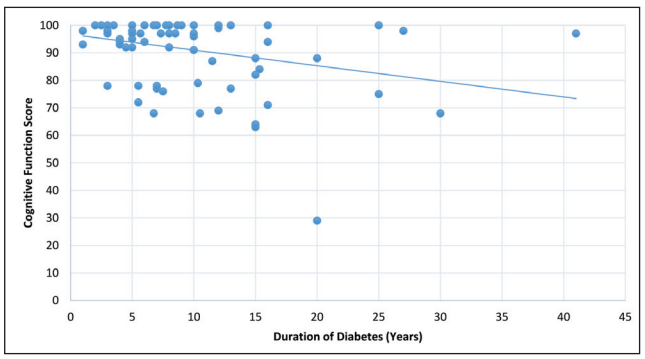

Figure 2: Relationship between the duration of diabetes and cognitive function

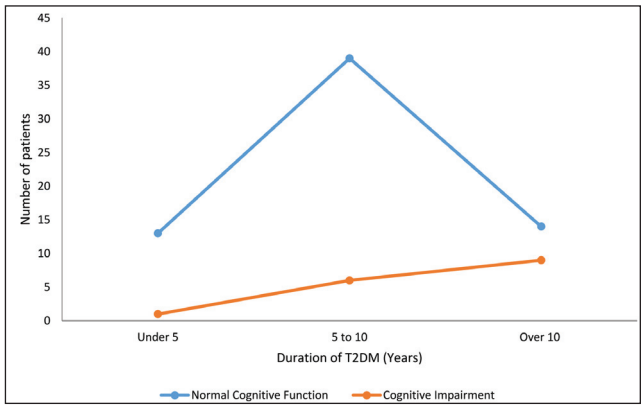

Figure 3: Effect of the duration of diabetes on cognitive function

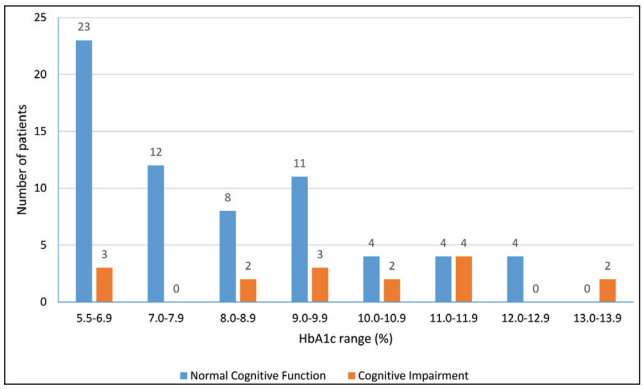

Figure 4: Frequency of cognitive function across the ranges of glycosylated hemoglobin

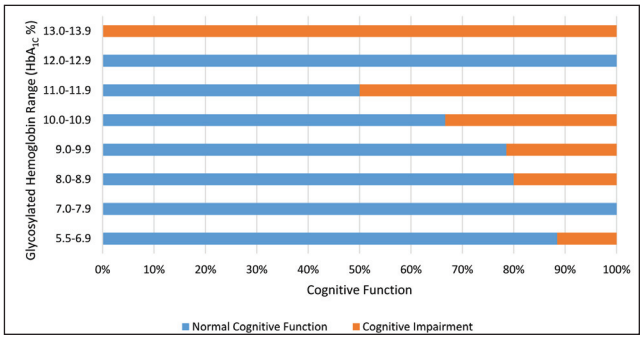

Figure 5: Level of glycemic control and cognitive function

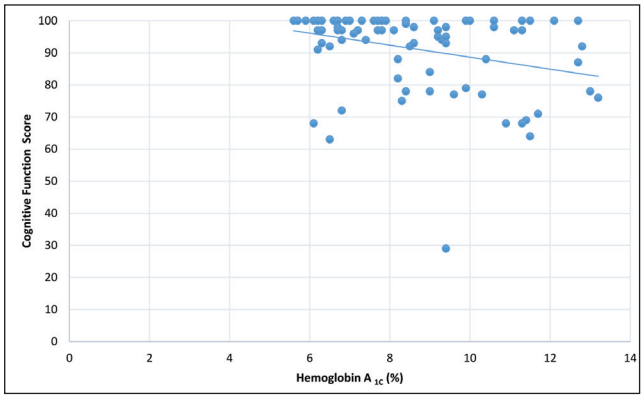

Figure 6: Relationship between the glycosylated hemoglobin level and cognitive function score

relationship between glycosylated hemoglobin level and the cognitive function.[2-8,16,17]

There are many factors that can explain this difference. First, age-related cognitive impairment is mostly reported after 60 years of age,[11] and about 10-20% of the healthy population above the age of 65 suffers from mild cognitive impairment.[12,13] In our study, the mean age of T2DM patients was 50 years, while the mean ages of the patients in all of the other studies were above 60; e.g., in the Action to Control Cardiovascular Risk in Diabetes-Memory in Diabetes (ACCORD-MIND) group study,[2] the study by Yaffe and colleagues,[4] and the study by Crane and colleagues,[16] the mean ages were 62.5 years, 74.1 years, and 74 years, respectively. It is, therefore, appropriate to state that a normal age-related natural decline in the cognitive function after the age of 60 years could have increased the likelihood of a higher prevalence of cognitive impairment in the studies that included older adult T2DM patients, resulting in a strong relationship between cognitive impairment and poor glycemic control in this population. In other words, poor glycemic control alone could not be fully responsible for the cognitive decline in the older adult patients. Second, structural brain imaging studies on T2DM patients over 60 years old show more pronounced micro- and macrovascular complications, such as lacunar infarcts, which do not tend to occur in younger adults.[6] Such vascular changes are associated with a higher prevalence of cognitive impairment and dementia in the older adults.[5,6]

Third, older adult patients tend to have more associated comorbid conditions compared to younger adults, such as hypertension, hyperlipidemia, atherosclerotic cardiovascular disease, depression, etc. These comorbid conditions are independently associated with cognitive decline.[18] Furthermore, in the ACCORD-MIND study, it was observed that in the older adult T2DM patients with high cardiovascular risk due to many of the comorbid conditions mentioned above, a tight glycemic control did not show a reduction in cognitive decline.[19,20]

The prevalence of cognitive impairment in our young adult T2DM patients was 19.5%. Although it has been reported that less than 4% of the healthy adults in the USA under the age of 65 years suffer from dementia,[14] the prevalence of cognitive impairment in the healthy population under the age of 60 years is not known. Many studies have reported 10-20% prevalence of cognitive impairment in the healthy population above the age of 65 years.[12,13,21] It would be appropriate to say that we

need further studies that would assess the prevalence of cognitive impairment in the healthy younger adult population under the age of 60 years in order to draw a comparative analysis of our findings.

We found a moderately negative relationship between the duration of T2DM and cognitive function scores. More than half of our patients with cognitive impairment had T2DM for more than 10 years. Our findings were similar to the observations of the Maastricht Aging Study,[22] which included patients who were of 40 years of age or older. The study showed that after 12 years, T2DM was associated with a decline in cognitive function, particularly in information-processing speed and executive function, compared with individuals without T2DM. Similar conclusions were drawn in the two Whitehall II studies of patients with a mean age of 55.6 years, which compared the Framingham general cardiovascular disease risk score and the Framingham stroke risk score with the Cardiovascular Risk Factors, Aging and Dementia (CAIDE) risk score.[23,24] The study found that after 10 years, T2DM was associated with decline in a global test of memory and reasoning. However, in the Diabetes Control and Complications Trial (DCCT), which included patients with the mean age 27 years and type 1 diabetes mellitus (T1DM), there was no decline in the cognitive function after an average of 18 years.[25] These findings indicate that factors other than the duration of diabetes (T1DM or T2DM) must be responsible for adverse influence on cognitive function.

We found that 50% of the patients with cognitive impairment were African-American patients. Our findings are consistent with the study conducted by Mayeda and colleagues, which showed that African-American patients with T2DM had a 41% greater annual decline in processing speed scores and 50% greater annual decline in verbal fluency scores than those without T2DM.[26]

We found that the level of education did not significantly affect the cognitive function in our young adult T2DM patients. Our findings differ from the findings of studies that showed significantly greater risk with elevated glycosylated hemoglobin levels in lower-educated adults than in higher-educated adults.[27]

Several limitations of our study deserve consideration. Baseline cognitive function scores were not available, which could have helped in comparing the effect of duration and level of glycosylated hemoglobin on the cognitive function over a time period. We had a limited sample size due to our strict selection criteria to include only young adult patients with T2DM. Although there are many modalities to study the effect of T2DM on cognitive function, such as neurocognitive testing, evoked response potentials, and magnetic resonance imaging,[28] there are limitations in administering a detailed neurocognitive test during routine patient visits in a primary care office due to time and cost. We chose to use the 3MS test, which incorporates four added test items and more graded scoring compared to the Mini-Mental State Examination (MMSE).[15,29] The 3MS test requires less time and is easier to administer during a routine office visit. The major strength of our study was establishing a broad range of exclusion criteria so as to exclude patients with any confounding comorbidities that might impact cognitive function.

We conclude that cognitive impairment affects one-fifth of young adult patients with T2DM and it is weakly negatively related to the glycosylated hemoglobin level, moderately negatively related to the duration of diabetes, and weakly positively related to the level of education.

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