Dyschromatopsia in Diabetes Mellitus and its Relation to Metabolic Control

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In 102 insulin-dependent diabetic patients without retinopathy and with visual acuity 20/20, the Farnsworth-Munsell 100-Hue test was performed, and glycosylated hemoglobin (GlHb) levels were determined. In 70% of the patients, a dyschromatopsia in the yellow-blue axis (tritanopia) was found. No correlation existed between duration of diabetes and tritanopia. On the other hand, the degree of this visual defect was positively correlated with GlHb levels. Thus, dyschromatopsia might be associated with poor metabolic control. It is suggested that dyschromatopsia is a consequence of hypoxia at the neuroepithelial level. The high levels of GlHb could be a contributory cause of hypoxia by reduction of both oxygen release capacity and erythrocyte deformability. DIABETES CARE 5: 375–378, JULY–AUGUST 1982.

A color vision defect has been reported to occur in diabetic patients.[1,2] Such an abnormality affects mainly the yellow-blue axis (tritan and/or tetartan deficiencies),[2–4] and, besides its theoretical relevance, it has practical implications for the Clinitest interpretation by diabetic patients performing home blood glucose monitoring.[5,6] This defect cannot be ascribed to diabetic retinopathy, inasmuch as it occurs also in retinopathy-free diabetic patients.[2,3]

In the present study an attempt was made to verify whether dyschromatopsia in diabetes is related to the duration of the disease and/or to the degree of metabolic control, assessed by determining glycosylated hemoglobin (GlHb) levels.

PATIENTS AND METHODS

One hundred and two subjects (45 men, 57 women) with insulin-dependent diabetes mellitus, aged 10–29 yr (mean 17.06 ± 5.23), have been studied. The duration of diabetes ranged from 0.2 to 13 yr (mean 4.46 ± 3.06). None of the female patients was on contraceptive pills. Patients with congenital protan and deutan defects, assessed by means of the Ishihara test,[7] were excluded from this study. All subjects were enrolled on the basis of both absence of retinopathy, assessed through ophthalmoscopy on dilated pupils, and visual acuity 20/20.

Among the 102 patients, five were recently diagnosed and therefore underwent their first examination of fundi; the others had been examined every 6 mo during the routine follow-up in our diabetic clinic.

In each patient the Farnsworth-Munsell 100-Hue test[8] was performed within a few days of examination of fundi. The errors made in putting the hues in the proper sequence were plotted on the Farnsworth-Munsell 100-Hue diagrams,[8] and the total score (TS) was calculated. The same day, GlHb (as $\%HbA_1$) was determined using Isolab's Fast Hemoglobin test system (Isolab, Inc., Akron, Ohio).[9] The significance of correlation coefficients was tested by one-sided Student's t test.

All ophthalmologic examinations were performed by A. S. and C. M., ophthalmologists.

RESULTS

Data on the whole group of diabetic patients, with mean and extreme values of TS and GlHb, are reported in Table 1.

Table 2 and Figure 1 show that in 72 of 102 cases the TS was higher than the upper limit of scores recorded in age-matched normal healthy subjects.[4] In all cases but one, including the subjects with normal TS, the errors clustered in the yellow-blue axis (tritan and/or tetartan) (Table 2).

Statistical analysis showed positive correlation ($r = 0.239$; $P < 0.01$) between TS and GlHb levels (Figure 2), while no correlation was found between TS and duration of diabetes.

TABLE 1
Data on age, duration of disease, total score of errors in the Farnsworth-Munsell 100-Hue test, and levels of glycosylated hemoglobin in the 102 diabetic patients

	Age (yr)	Duration of disease (yr)	Total score	GlHb (%)
Mean	17.06	4.46	96.74	9.65
SD	5.23	3.06	69.57	3.54
Minimum	10	0.2	8	3.8
Maximum	29	13	444	19
Range	19	12.8	436	15.2

TABLE 2
Total score of errors in the Farnsworth-Munsell 100-Hue test and color discrimination in the whole group of diabetic patients

	Blue-yellow axis	Red-green axis	Normal	Total
Normal total score	19*	1	10	30
Pathologic total score	72	—	—	72
Total	91	1	10	102

* Figures refer to number of subjects.

DISCUSSION

In this study, as in several others,[2-4] a yellow-blue dyschromatopsia is present in 70% of insulin-dependent diabetic patients. This abnormality is very rare as a primary defect (congenital tritanopia). On the other hand, it is typically caused by several pathologic conditions (acquired tritanopia).[4]

Tritanopia has been described in megaloblastic anemia, [10] in carotid occlusive disease and chronic central retinal vein occlusion,[11] after inhalation of a reduced oxygen gas mixture,[12] and in beta-thalassemia.[13] The common denominator for yellow-blue defects caused by such diseases, or by drugs,[14,15] seems to be decreased oxygen uptake by the neuroepithelium with consequent receptor damage.[16] However, the pathogenesis of dyschromatopsia in diabetes is still unclear.

In our patients no correlation exists between dyschromatopsia and duration of diabetes. Moreover, the defect is found in diabetic patients without retinopathy. Taken together, these facts suggest that tritanopia is not a consequence of an early retinopathy. The correlation found between GlHb and yellow-blue deficiency suggests, on the other hand, that dyschromatopsia is related to the degree of metabolic control.

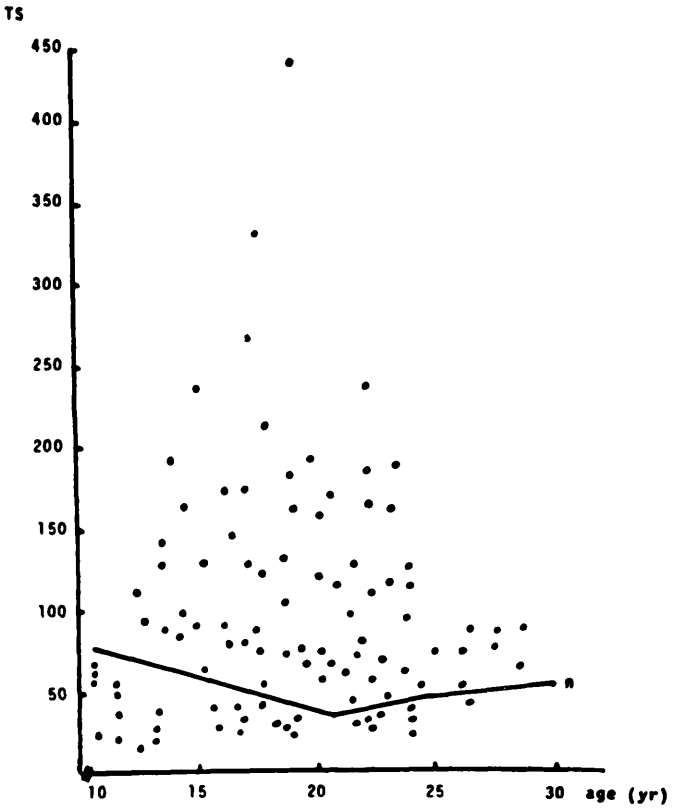

FIG. 1. Total score (TS) values of the 102 subjects. The solid line (n) indicates the upper limit of scores recorded in age-matched normal healthy subjects by Verriest.[4]

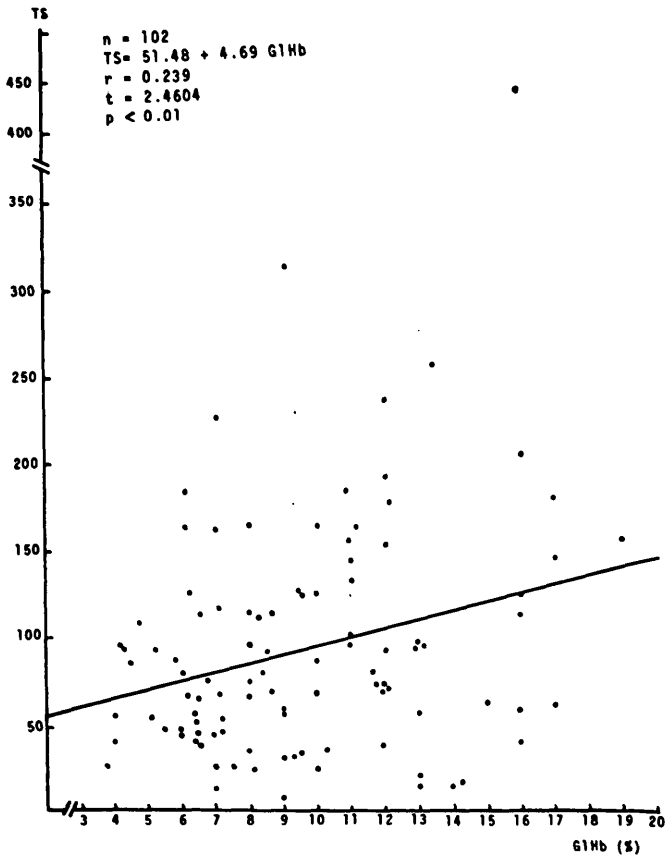

FIG. 2. Total Score (TS) values related to glycohemoglobin (GlHb) levels in the 102 subjects.

How this visual defect is produced by poor metabolic control in unknown. We suggest two hypotheses. According to the first, the defect might be seen as a consequence of diabetic neuropathy affecting the optical nerve. In the second hypothesis, on the other hand, the dyschromatopsia might stem from hypoxia at the neuroepithelial level, in agreement with Lagerlöf's concept.[16]

Regarding the first hypothesis, it should be kept in mind that generally optic nerve diseases cause defects in the red-green axis.[4] However, Ohta[17] states that pathologic changes between the optic disc and chiasm can also result in yellow-blue defects. Therefore, the possibility that such an abnormality represents an early manifestation of diabetic neuropathy cannot be ruled out.

In spite of this, the hypothesis of a hypoxic mechanism is, in our opinion, more tenable, chiefly because the same type of dyschromatopsia is seen in a variety of conditions in which hypoxia is the only common feature.

If this is the case, the high GlHb levels could be both an indicator of poor metabolic control in the last 30,[18–25] or 7 days,[26] and a pathogenic link between diabetes and dyschromatopsia. In fact, glycosylated hemoglobins can lead to tissue hypoxia through both increased oxygen affinity[27,28] and reduced erythrocyte deformability.[29] For the moment, however, the relationship between diabetes and tritanopia remains poorly understood.

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